VOLUME 20 July, 1954 NUMBER 7



# THE AMERICAN SURGEON



PUBLISHED MONTHLY BY
THE WILLIAMS & WILKINS COMPANY
BALTIMORE 2, MARYLAND

Entered at the Post Office in Baltimore, Maryland as second-class matter.

CONTENTS, JULY

# The American Surgeon

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*Subscriptions. The American Surgeon* is published monthly, and twelve issues in a calendar year constitute a volume. Subscriptions are sold on a volume basis.

Subscription price in the United States and countries in the postal union: $10.00 per volume. In countries outside the postal union: $11.50. Single copies, when available, $1.00.

*Publisher*

THE WILLIAMS & WILKINS CO.

Mt. Royal & Guilford Aves., Baltimore 2, Md.

THE
# AMERICAN SURGEON

Vol. 20, No. 7 July, 1954

## THE VALUE OF A CANCER CLINIC IN A COMMUNITY HOSPITAL

J. R. YOUNG, M.D.

*Anderson, S. C.*

Before beginning my president's address, I have this word for the group of young men who have joined the Congress during the year. Speaking for the entire membership, I wish to assure each of you that we are happy to have you join ranks with us. We believe that each succeeding company of recruits brings to our organization a useful leaven of enthusiasm that rightfully belongs to the well trained young surgeon of today. The postgraduate training which many of you have received in the principles and practice of surgery is far superior to that received by many of us. Indeed we believe your training has been the best the world has ever had to offer, and we not only congratulate you but would also like to congratulate those members of our profession who give so freely of their time and energy to medical education. They are doing great work and merit the appreciation and support of the entire profession and of the public.

It is fitting that you young men who have cast your lot with us be told something about the organization you are joining. The Southeastern Surgical Congress was conceived about 25 years ago and held its first annual meeting 23 years ago. The sole purpose of the organization was to help raise the standards of surgery in the southeastern portion of the United States. Each of you has been proposed as a Fellow in the Congress by the members in your respective states because they deemed you a well-trained, reputable surgeon who would practice the art and science of healing in accordance with the ethical standards of our profession. The Southeastern Surgical Congress might sum up its view on medical ethics in this way:

1. We believe that we owe it to ourselves and to our patients to render them the finest service of which we are capable.

Presidential address presented during the Birmingham Assembly of The Southeastern Surgical Congress, Birmingham, Alabama, March 8–11, 1954.
Director of the Rose E. Ramer Cancer Clinic, Anderson, S. C.

2. We believe this service should be rendered well seasoned with kindliness, based on a broad understanding of the weaknesses and foibles of human nature.

3. We believe that in addition to having this high obligation to our patients, we likewise have a very genuine obligation to our profession. It should not be ours alone to enjoy the privileges of our profession, but it should be our daily endeavor to so conduct our affairs that the honor and reputation of our profession would be enhanced.

4. We also believe that as physicians we have a definite obligation to the well people of our respective communities. We believe that each of us should consider himself a watchman and do all in his power through appropriate voluntary and state health agencies to acquaint his people in health matters.

5. Aside from these professional duties which are ours, we believe that each of us owes to church and state the unswerving loyalty of a grateful citizen.

The Southeastern Surgical Congress is not an exclusive organization which ascribes to its charter members any superior moral excellency or wisdom. Rather is it our desire to be inclusive and to number on our rolls every well-trained surgeon in the southeast who is honest and ethical and is practicing his high calling with due regard for the service he may render his profession and his patients.

Nor does our organization view with alarm any dire threat of ghost surgery, fee splitting or similar bastard practice. Such evils have been practiced through all the history of medicine by an exceedingly small minority of doctors; but we believe that the vast majority—99.44 per cent, maybe—of our members are innocent of such conduct. You were proposed for membership in the Congress by the Fellows in your state because they thought that your moral stamina and ethical concepts would immunize you against such conduct. For we believe that if a man does not have within himself the makings of a gentleman no star chamber edict or mouthy muck raking will cause him to change his pattern of behavior. The Ethiopian's skin and the leopard's spots are permanent decorations.

Down the years as you young men propose others for Fellowship in the Congress, may we request that you rate integrity as first on the list of requisites. Surgical skills may be acquired, but moral stamina is from within, and if this is not possessed by a surgeon we believe he will not feel at home in the climate of the Southeastern Surgical Congress.

Our composite experience has been that the annual meetings of the Congress have served remarkably well in bringing us up to date on many problems in surgery. We believe you will not find a better program at any meeting you attend. And we commend to you very highly the annual meetings of the Congress as furnishing the best available means of reviewing in a short time a wide range of surgical problems.

As members of the Congress you will receive each month the American Surgeon. In this well edited journal you will find rewarding reading and you will look forward to its regular visits with pleasure.

One of the privileges of the members of the Congress is to listen during each annual meeting to an address by the president. Having now been duly received into full fellowship of the Congress, you are hereby sentenced to listen to this address by your president.

Since the meeting of the Congress in Atlanta two years ago, when I was honored by being made president-elect of the Southeastern Surgical Congress, I have thought many times on the nature of the message which it would be my privilege and duty to deliver on this occasion. It does not seem the proper time to present just another paper on a surgical subject so, following the example of many illustrious predecessors, I propose a rather philosophical discussion of a subject that I believe will be of interest to all of us.

It has been my good fortune to have witnessed the marvelous changes that have come about in surgery for about half a century. Beginning in the horse and buggy days, before hospital facilities were available in my town, we operated upon the patient on the kitchen table by the light of the kerosene lamp. I am sure I could relate an interesting tale from those horse and buggy days up to this good year of 1954, but after the tale were told no one would be any better prepared for the problems that confront us today. Therefore, our eyes and thoughts will not be turned backward, but to the current problems of today and the future.

In the background of all that I shall have to say there will be the thought of the changed rating which the average Mr. Citizen has of the present day doctor, as compared to his appraisal of the doctor in the horse and buggy days. We sometimes embellish the story of our immediate professional forebears with not a little tradition that may be more romantic than it is factual. We ascribe to them credit for having first devloped in the minds and hearts of their contemporaries a very high esteem for the members of our profession. This is by no means true. History teaches that this high degree of esteem and affection which we may enjoy has been shown by many peoples for many centuries. Its fervor has shown something of a pendulum-like regression and resurgence. In the book of Ecclesiasticus, which is one of the books of the Apocrypha and was written some 200 years before the birth of Christ, we find an author by the name of Jeshua giving voice to this arresting poem:

> "Honour the physician with the honour due unto him;
> For verily the Lord hath created him;
> For from the Most High cometh healing;
> And he shall be honoured even by the King;
> The skill of the physician shall lift up his head;
> And in the sight of great men he shall be exalted.
>
> "The Lord created medicine out of the earth,
> That He might be glorified in his marvelous works:
> And he that is wise will not despise them.
> With them doth the physician heal a man,
> And taketh away his pain.
> Yea, there is a time when in his hand is the issue of life;
> For by his skill doth he make supplication unto the Lord;
> That He may prosper him in giving relief
> And send healing for the maintenance of life,
> And from him is peace upon the face of the earth."

We see from this beautiful poem that the affectionate esteem held for the

physician of that day was very similar to that enjoyed by our more recent forebears. During the intervening centuries the varying change in rating has been more quantitative than qualitative, and the flavor and fervor of this esteem in successive generations has largely varied in direct proportion to the skill and dedication of the physicians of each generation.

In the long ago a very picturesque statement was made by a vivid author; "So thou, O son of man, I have set thee a watchman unto the house of Israel; therefore thou shalt hear the word at my mouth." Please remember that I am speaking to you as the watchman whom you selected and not as a private individual. Were I speaking in that capacity the personal pronoun would not be so much in evidence, nor would I speak for the Congress in the tone of one having authority.

The subject on which I wish to drape my thoughts is *The Value of a Cancer Clinic in a Community Hospital*. In order that we may be sure we are all thinking in the same terms some definition is necessary. By a cancer clinic we mean an organized group of doctors and associates selected from the staff of a hospital who, at a regular time and place, meet for the examination of patients who are thought to have cancer, and who further are prepared and equipped to treat those patients who are found to have this disease. An approved clinic must have efficient secretarial help, as well as a satisfactory follow-up system. The definition which I have in mind of a community hospital is one that is not a large teaching hospital, but is a hospital built after some acceptable pattern by the citizens of the community. The staff of the clinic must include at least a competent pathologist, a competent and equipped radiologist, and one or more surgeons trained to do radical cancer surgery. It is my opinion that a cancer clinic so manned and equipped is capable of rendering a very definite worthwhile service.

The first and very obvious value of such a cancer clinic is to the patients treated. It is undoubtedly true that many cancer patients can be brought to a local clinic who would not consent to go to a more distant hospital. We believe that the people who live in the states covered by the Southeastern Surgical Congress would be better served by such regional cancer clinics than they would by larger clinics conducted on a state level. We have been working in such a clinic in our hospital in Anderson for the past 14 years, and we have seen many cancer patients relieved who would certainly have died had not the facilities of this clinic been available. We have now on the rolls of our clinic 187 patients who have been cured, or in whom disease appears to be under control. We have 196 other more recent patients, most of whom are making satisfactory progress. Three hundred and forty-three of our cancer patients have died during this 14 year period. During the last illness of these patients our field worker was able to render an appreciated service to the patient and family.

But probably an even greater value that the cancer clinic may render is in the field of education. It is certainly true that in most communities the composite thought of its citizens is that cancer is usually rapidly fatal and that operation only hastens the fatal outcome. This opinion, inherited from countless generations, seems now imbedded in the cellular protoplasm of mankind. To uproot

or wholly loose such deeply ingrained opinion must necessarily be a gradual process. We believe that a well-conducted cancer clinic may serve as a valuable aid in accomplishing this end. Abstract teaching in regard to the curability of cancer, as contained in many available pamphlets, is not nearly so convincing to the average person as the living presence of a neighbor or acquaintance who has been relieved of cancer. By far the most effective sermon on the curability of cancer is preached from the reclaimed patient as a text. In this way the cancer clinic may serve as a catalytic agent in dissolving the superstition and well-nigh fatalistic attitude that is so widespread.

Another benefit which a cancer clinic such as outlined above may render in a community hospital is in keeping all the members of the staff alerted in regard to cancer. One or more times a year the entire hospital staff should have the benefit of reviewing the accumulating experience of the cancer clinic. At frequent staff meetings gross specimens and microscopic slides should be presented by the pathologist. A cancer clinic satisfactorily staffed and equipped and conducted as we have described may become the outstanding service unit in a community hospital. The service will extend not only to the patients of the clinic, but will very effectively and progressively serve the community as a warning or alerting agency, in regard to cancer. The value of the cancer clinic as a professional teaching agency will vary in direct proportion to the use of the clinic's consultation service by all the members of the hospital staff.

But all of us who conduct cancer clinics know that many, far too many, patients brought to the clinic are found to be hopeless as far as cure is concerned. While this is true, probably no department in the hospital is so strategically situated to impress the patient and his family that all the resources of the hospital are available to ameliorate his disease. These are the cases in which palliative surgery and radiation therapy is often times indicated and where hormones and other palliative therapeutic agents may be used to advantage. It is usually not possible, and probably not desirable, that the patient be kept in the hospital for long periods of time; but by careful follow-up care, frequent visits by the patient to the clinic and visits to the home by the field nurse, the patient will become convinced that the resources of the hospital are dedicated to his care, and a natural feeling of appreciation will follow. If the attitude of the cancer clinic patient and his family is not one of grateful appreciation, there is probably something wrong in the conduct of the clinic. It may be, however, that this debit item is due, not to the personnel of the cancer clinic itself, but to an unfriendly atmosphere that prevails in the hospital where the clinic is situated. We know of no reason why the personnel of the hospital should be unfriendly or lacking in sympathy for the cancer clinic patient. In my own state, and in many other states, the Cancer Division of the State Board of Health makes satisfactory financial arrangements with the hospitals where the clinics are situated. Truth compels us to say that the lack of the friendly atmosphere may be due to the members of our own profession more than it is to other members of the hospital personnel. This disapproval, or neutral reaction to the activities of the cancer clinic on the part of some members of our own profession, stems from the phobia

which exists in the minds of many members of our profession towards Socialized Medicine. We are well-nigh unanimous in our opposition to this pattern of medical practice in our country, but there is a large difference of opinion as to the best method of preventing Socialized Medicine from becoming the accepted pattern of practice. After a half-century's experience in medicine, it is my humble opinion that the surest way of preventing this from ever happening would be for the doctors in each community to furnish competent medical care to all its citizens! This, of course, is a big order, but I believe it can be done. It cannot be done after the pattern of the horse and buggy days of seeing each patient in his home; but with good roads, easy transportion, and closely spaced hospitals, the health needs of our citizens can be satisfactorily met, provided the doctors cooperate and organize to supply this need.

Now it has been found that cancer is such a major problem in our national life (one out of each five of us at the present incident rate being destined to develop this disease) that it can best be handled by setting up clinics in connection with hospitals, where patients may be cared for regardless of their inability to pay for this service. The close cooperation between the state board of health, federal health agencies, and voluntary health agencies, such as the American Cancer Society through its divisions, have made it possible for cancer clinics to be established throughout our country. There are now in the southeastern part of the United States 92 such clinics that are rated as competent. I believe that each of these clinics is earnestly attempting to render a worthwhile service to its patients. A more widespread endorsement, support and use of these facilities by the medical profession would not only be in the interest of the low-income patients themselves, but would be a very eloquent demonstration to the public that the medical profession accepts the challenge of caring for medically indigent cancer patients. Now if a satisfactory job can be done in the very major problem of cancer, it would seem reasonable to suppose that all of the health problems of our citizens might be met, were similar organized and determined efforts made to that end.

But the consultation service of the cancer clinic should be available, not only to the cancer clinic patient, but to every patient with a malignancy who is admitted to the hospital and to every case that occurs in the area of the clinic. It is really in this class of private patients who are victims of cancer that the cancer clinic may do its most valuable service in the realm of public relations. The pattern for rendering this service should be somewhat as follows: in order to increase the frequency and value of such consultation service the clinic staff should agree that the service is available at little cost; to many low-income private patients we think no fee should be charged. When this service becomes routine, consultation will be somewhat as follows: on request from the physician in charge, the proper individual from the cancer clinic staff will conduct the consultation. He will become thoroughly familiar with the clinical findings of the case, and then he will see the patient, with the physician, and himself make a careful appraisal of both the patient and his disease. Then, after a frank discussion, the clinic consultant will, as much as conditions will warrant, congratu-

late the patient and his family upon the thorough way his physician has handled the case. It will always help the patient and his family to be told they have exercised good judgement in their selection of a private physician, and such technic never offends the physician in charge. The consultant should then emphasize to the patient and his family the encouraging aspects of the case and then impress upon them that the treatment advised is not only thought to be wise, but that it is the concensus of medical opinion of the world that this treatment is the best now available anywhere. When the clinic functions in this way, all the members of the hospital staff, together with the members of the cancer clinic staff, may confidently assure the patient that all the resources of the hospital are at his command and that no effort will be spared. If such sympathetic atmosphere prevails during the stay in the hospital, it will serve very effectively as a shock-absorber when the time arrives for making financial settlement at the close of a hospital stay. Much of the present criticism of hospitals and doctors stems from this belief held by many people that we doctors send to the hospital many patients who might recover satisfactorily at home, and they further believe that the cost for this hospital and medical care is far too high. We will not argue too much as to the validity of this opinion held by so many. Suffice it to say that the increased cost of hospital care is due to very demonstrable causes; first, the increased cost of commodities and services which the hospital furnishes; second, the very marked increase in the scope and skill of services. While it is true that the annual income of physicians and surgeons has increased, as has the income of everybody else, most of the increase in the income of the surgeon has been due to the fact that more people have been able to pay his regular fee than in former years. There has been comparatively little change in the fee schedule for professional services rendered in most communities.

However, it is true that for the proper care of a cancer patient, few would argue that home care is superior or equal to treatment that may be received in a well equipped hospital. So there is usually no resentment on the part of the patient or his family that the cancer patient was advised to go to the hospital. However, there is always some resentment on the part of the average citizen against being sick at all. He has put little in his family budget for major illnesses, and many of the insurance policies that satisfactorily cover minor illnesses are entirely inadequate for the major cost of cancer treatment. It is therefore a very auspicious thing to have a prearranged, smoothly functioning shock-absorber available for this financial transaction. Nothing will serve so well in this capacity as a very definite impression on the part of the patient and his family that the entire resources of the hospital have been available for the physician in charge of this case and that regardless of the amount of his hospital bill he has experienced value received. This quirk of human nature is an interesting thing to observe. The afferent dollar to the average person is a very insignificant coin—maybe like one of the widow's proverbial mites. He thinks that for the service he has rendered he should receive far more of the said coins. However, the efferent dollar, which may have remained in his possession for only a short while, by some strange alchemy has enhanced in value and looks as big as a cart wheel, and, as he sees

it depart, he cannot understand why a couple of them should not pay for almost any commodity or service. Now it could be that the members of our own profession have developed a slight touch of this malady! I believe it is undoubtedly true that no small proportion of the resentment that exists in the minds of many people towards the members of our profession is closely associated with the item of folding money. Nothing would come as near dispelling this belief as would the habitual effort of every doctor to follow the advice of Sir William Osler, to give every patient more than he charged them for. The human male, as well as female, is pleased to think that he received bargain-counter value. Was not at least part of the esteem and affection, and even veneration, given to the family physician of the old school due to the fact that the patient's family so often felt that they were getting in kindly service far more than they were paying for? And, between ourselves, it could have been that some of the second-mile technic adopted by our illustrious forebears was due to the vicissitudes of travel, roads, and weather. Be that as it may, the fact remains that according to legend and letters doctors of the old school were more highly esteemed than we are. While the doctors of today may not use the same second-mile technic as did our forebears in the horse and buggy days, the same second-mile principle might be very convincingly used by us now if we habitually followed the advice given by Osler. It should therefore be the solemn duty of each one of us to remember that the financial transaction incident to the service we render our patients may be so conducted as to offend the sense of justice and fairness of the patient. And as a result the reputation of our entire profession suffers. Far be it from me to presume to offer an original formula for this procedure. However, I believe that the majority of the members of the Southeastern Surgical Congress have found that the Open Sesame to a mutually happy handling of this matter is always present when the advice of the Great Physician, as summed up in the Golden Rule, is made to season this transaction. The physician who is a disciple of the Great Physician and uses this Golden Rule in dealing with his patients will not only endear himself to them but will do much—very much—in restoring to our profession the esteem and affection of a grateful people.

# NERVE AND TENDON REPAIR OF THE HAND

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As in surgery anywhere, surgery of the hand depends, or is based upon two major considerations: indications for surgical intervention and the technic of carrying it out. If either is at fault, the end result is failure. Nowhere is this so forcibly true as in the surgical treatment of nerve and tendon injuries of the hand, whether this be in the care of the acute cases or in the secondary surgical management of hand injuries.

One of the real problems which the surgeon must solve when confronted with an acute injury is whether or not he is justified in carrying out primary nerve and tendon repair. This is not always an easy question to answer, nor is it always easy for the surgeon to accept the logical conclusion of his deliberation. The presence of divided nerve and tendon ends within the wound would seem to demand their repair, but there are a number of circumstances which council against it. Many factors may be favorable, but it is the unfavorable factors which count, and one such contraindication will outweigh any number of favorable ones.

Ben Rank of Australia very sensibly has divided wounds of the hand into two categories, a subdivision which is of extremely practical value for the surgeon. One group of wounds is called the *untidy* injury. This group includes the crushing, tearing, macerating injuries with extensive tissue destruction and loss; traumatic amputations; crushed bones and deep abrasions in which are found open fractures; loss of or devitalization of skin; joint injuries, disruptions, crushed muscle, torn and frayed nerves and tendons, and grinding in of dirt, grease and other foreign material. In injuries of this type, the problem confronting the surgeon is extremely complex. He first must cleanse and rid the hand of all foreign material and hopelessly devitalized tissue; and, from what is left—often seriously damaged and with lowered vitality—he must plan largely to reconstruct only the foundation of a hand and secure adequate skin coverage by whatever means are feasible or possible. Fine, detailed reconstruction and niceties of repair usually must be subordinated to the bare essentials of securing healing with the hand or its remnants in the position of function; its skeleton restored and the part covered with skin. In wounds of this type, nerve repair often may be done, providing it is not necessary to open up undamaged tissue to secure retracted ends. Tendon repair, however, is practically never possible except that occasionally extensor tendon ends on the dorsum of metacarpus and digits may be approximated with fine silk sutures.

These—the *untidy* injuries—therefore, form a group in which the surgeon, who sees the patient initially, realizes that nerve repair occasionally may be done; tendon repair practically never.

* Read during the Southeastern Surgical Congress, General Assembly, March 8–11, 1954, Birmingham, Ala.

The other type of injury—the *tidy* injury—as Rank has called it, is caused by some sharp-edged instrument, cutting cleanly through the tissues of the hand and causing little damage beyond that of its actual wound tract. Such wounds due to glass, knives, steel tools or tin plates, may cause very extensive nerve and tendon damage, but the surrounding tissues are not traumatized, although there may be disturbance of blood supply in flaps of skin raised up by the traumatizing agent. It is in wounds of this type that the surgeon must decide whether or not he should attempt repair of divided nerves and tendons.

Decision on feasibility of nerve and tendon repair in these cases is made on three primary considerations. One of these is the time which has elapsed since the injury; a second is the condition of the wound as regards the presence of, or threatened development of wound infection; and the third is the facility, including the technical skill of the operator for carrying out indicated repair.

At the outset, it may be stated categorically that if, for any reason, the surgeon believes that he cannot anticipate primary healing of the wound, he should not attempt tendon repair. He obviously should inform the patient relative to his decision and the reasons for it, but should feel no compunction to attempt repair under unfavorable conditions; nor should he receive censure for failing to undertake repair in such cases. Success of tendon repair first of all depends upon securing healing by primary intention.

The question of time is somewhat difficult to evaluate today, due probably to two factors: In the early days of tendon surgery—and by *early* I mean the nineteen hundred twenties and thirties—tendon repairs on the hand were so fraught with danger, that they were never attempted in wounds over four hours old, and often not done in wounds over two hours old. These time limits, born of painful, practical experience, have been difficult to re-evaluate in the light of modern surgical advances, more particularly the antibiotics. All surgical experience today is colored by the use of antibiotics, and, oftentimes, to the neglect of cleanly surgical care. The antibiotics well may have extended the original safe limits for primary tendon surgery, although it must be remembered that there has been much improvement in the care of wounds in general; a greater appreciation of the factors leading to wound infection, particularly the significance of devitalized tissues in leading to disturbed healing. Likewise, surgical technic has improved; tissues are handled with greater gentleness, and the surgeon does not so often leave in his wake great areas of devitalized tissues and coarse suture material. Flynn's report of successful suture after the expiration of our previously determined safe time limits indicates what may be done by careful technic in a well controlled surgical service. However, it must be noted in his series that the vast majority of his cases were seen eight hours or under following inception. The discussion regarding time limits concerns only the first 12 hours following injury; after this time there are few, if any surgeons experienced in this work who advocate primary repair.

But time is only one factor which must be taken into consideration. Of more importance is the question of wound contamination, and this is often difficult to evaluate since one does not always know what the possible sources of contamina-

tion have been. The accident, itself, may have been caused by a seriously contaminated object, or virulent contaminants may have been introduced into a wound following the injury. Protection of the wound starts at once after wounding by the application of an adequate first aid dressing. Each time the wound is uncovered, it is vulnerable to further contamination. A careful chronologic history of the injury and its care until the patient is admitted for treatment is essential to a valid conclusion as to probable degree of wound contamination. If, after consideration, the surgeon believes that the wound contamination is very likely to lead to wound infection, particularly if further trauma is added to the tissues, then primary tendon repair is not justified.

Occasionally, nerve ends present themselves in wounds which would not permit exploration for retracted tendons. It is permissible to suture the nerves under these circumstances, providing it can be done with a minimum of disturbance of the tissues. If there is hesitancy about formal suture, it would be well at least to approximate divided nerve ends with 1 or 2 fine silk sutures, passing only through perineurium to prevent retraction and to facilitate secondary repair.

Lastly, the surgeon should consider whether or not the facilities are available for repair and whether or not he is technically capable of carrying it out. The proper instruments and suture material should be available; facilities for anesthesia and at least one trained assistant should be present, since it is practically impossible to do these procedures without help. Above all, the surgeon, himself, should be sufficiently trained in the technic of nerve and tendon repair to do the procedures. Nothing is gained, and very much is lost, if a tendon repair is done and fails because of obvious faults in technic.

Some of the faults in technic merit special mention, since they may make secondary repair extremely difficult, if not impossible. The most frequent gross technical error made is the midline incision in a finger, palm or wrist to recover the retracted tendon ends. The scar always contracts, leading to flexion deformity, and usually is adherent over its entire length to the underlying area of tendon repair. Oftentimes, in the digit, the fibrous tendon sheath will be found to have been divided, and the whole anterior surface of the finger, including the tendon, fused into a solid scar. The situation is not so bad when the palm and wrist are involved, but it is serious enough. It is often impossible to correct the scar contracture satisfactorily, and secondary repair is seriously handicapped in the palm and wrist.

Incisions for uncovering retracted tendon ends in order to do repair must not only afford adequate exposure, but must avoid leaving scars crossing skin creases at right angles.

Another common error in technic is the use of heavy suture material for repairing nerves and tendons. While it is universally agreed that the material should be fine and nonabsorbable—silk, cotton, steel—it is not so universally understood what is meant by *fine* sutures. Tendon should never require sutures heavier than no. 0000 to no. 000000. My own preference is for no. 000000 twisted, untreated white silk. I have given up the use of any heavier silk. For repair of

nerves, and for securing final approximation of tendon ends, no. 000000 or no. 0000000 silk swaged on to a fine, sharp needle—arterial or nerve silk—is used.

The multitudinous details of nerve and tendon repair; the necessity of working in a bloodless field; careful hemostasis and closure without tension plus many other seemingly self-understood minutiae scarcely can be elaborated upon in this paper. They however must be mastered if one hopes to secure satisfactory results in surgery of the hand. Back of all of this technic lies the principle of gentleness in handling tissues—the *atraumatic technic* as Bunnell has so aptly called it. Until the surgeon learns to handle tissues as if they were the living, sensitive, easily-damaged substance that they are, he will never secure satisfactory functional results with nerve and tendon repair. The aseptic conscience must be supplemented by the atraumatic conscience; the surgeon must become just as aware of faults in handling tissues as in violation of asepsis.

In postoperative care, which the surgeon should know as well as operative technic, the fault infrequently not observed is the failure to immobilize tendons long enough after repair. Tendons require a minimum of three weeks to become sufficiently strong to permit motion, and another one to two weeks of protection against too active or strenuous use. In other words, the repaired tendon must be kept splinted in relaxation for three weeks after suture, and the patient should wear some sort of restraining apparatus for another one to two weeks while being permitted to use his hand. While it is motion we are seeking in the repaired tendon, use during healing simply leads to increased reaction, separation of tendon ends, adhesions and other wound disturbances which ruin the end result.

There are some differences of opinion in regard to the repair of flexor tendons in the digits. The difference concerns the policy to follow in the care of division both profundus and sublimis tendons over the proximal phalanx. Some contend that such an injury should never be repaired primarily, but simply closed and repaired by tendon graft later. It is my belief that, if the case is very favorable as regards contamination; is seen very early, and is a clean-cut wound without crushing or tearing of tissues; primary suture of the profundus may be done at the same time the sublimis is removed and the dense fibrous sheath over the site of tendon repair is excised. If ideal conditions do not prevail, it is my practice to close the wound and do a secondary repair by means of a graft three to five weeks later, or as soon as the tissues of the finger have become sufficiently soft to permit surgery.

Where proper conditions for primary tendon and nerve repair do not obtain at the time the patient is first seen following injury, the wound is simply cleansed; devitalized tissue is excised and primary closure made unless the wound is seen too late even for primary closure. Secondary repair of these injuries then is undertaken as soon as the tissues will permit. In instances in which primary healing has occurred, the tissues often will be sufficiently soft in three to five weeks. In some instances, a much longer period of time will be required and it sometimes may be necessary to give a course of physical therapy and tension splinting, and to do arthroplasties to mobilize digits before it is permissible to repair the tendons. It is not possible to go into the details of secondary repair, the various

indications for tendon grafting or tendon suture and lines of incision. Suffice it to say that before tendon repair is undertaken, the tissues of the hand must be soft and pliable and without induration, and the joints must be mobilized since the newly repaired tendon cannot be expected to mobilize them.

RESUME

Primary nerve and tendon repair on the hand may be undertaken with a good chance of success provided the patient is seen reasonably early—two to eight hours following his injury—if the wound is not seriously contaminated, and, if the surgeon possesses the skill to carry out the procedure and has the proper facilities for doing so. If these conditions do not obtain, tendon repair is best postponed and done as a secondary repair. Divided nerves often may be repaired in wounds in which tendons are not sutured, since exposure of nerve ends does not entail the wide dissection and exposure demanded in the mobilization of tendons. Proper technic is more than simply following the mechanical details of a procedure. It is a technic of gentle handling of tissues based upon an appreciation that living tissues are delicate and sensitive.

# THE MANAGEMENT OF MAXILLOFACIAL INJURIES

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Until recent years the treatment of fractures of the facial bones was considered a complex problem. This was not simplified by the number of contradictory procedures devised for the management of such injuries. Plastic surgeons, general surgeons, otolaryngologists and dentists developed methods of treatment, and opinions were never entirely in accord. In recent years there has been an increase in such injuries, resulting in part from the increased demand for speed and power in modern automobiles. Paralleling this increase the treatment of facial fractures has become less complex, possibly because of the realization that simpler methods give equally good results.

The object of this paper is to present a simple but satisfactory method of at least one acceptable form of treatment for the more common fractures and their related soft tissue injuries. By no means is it intended that these methods are the only acceptable ones, but we believe that satisfactory results can be obtained by such methods.

Frequently one hears of the *dental approach* or the *otorhinolaryngologic approcah* to the care of injuries of facial bones. There should be no competition between those interested in these injuries. There can be no doubt that close collaboration between the plastic surgeon, the otolaryngologist and the dentist serves the best interest of the patient with injuries of the facial bones.

Although the face must be considered as a unit, for the purpose of this discussion the various injuries may be divided into those of the nose, middle third of the face, and mandible. However, it constantly should be kept in mind that the treatment must be arranged to care for the fractured bones as a structural unit.

The goal to be sought in the treatment of these injuries is restoration of the occlusion of the teeth, function of the jaws, symmetry of facial contour and diminution of facial scars. There is no doubt that many facial deformities and subsequent operations can be averted if more time, thought, and care are devoted to the original injury.

Unless associated with a fracture of the ethmoid bone or the skull, severe injuries to the facial bones alone are not usually lethal. The first thought in the care of the patient should not be the injuries of the bones, but the patient's general condition. Immediate treatment should consist in diagnosis and management of shock, hemorrhage and of the associated wounds of the body which are so commonly seen. *The care of the fractured facial bones is never urgent at the expense of the general condition of the patient.* As a matter of fact, in most cases, except mandibular fractures, definitive treatment is given 5 to 10 days following injury.

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Presented during the Birmingham Assembly of The Southeastern Surgical Congress, March 8–11, 1954, Birmingham, Ala.

While the management of some fractured bones is better deferred for a time, wounds of the soft tissues should be treated within a few hours of the accident, if the general condition of the patient will tolerate it. Details regarding the care of soft tissue injuries of the face will not be given here, but brief mention will be made.[4]

A case presented below will demonstrate a method of management wherein treatment of the bone and soft tissue injuries could and should be carried out immediately and simultaneously. Such instances are not too common in the middle third of the face, but frequently are seen when one is dealing with fractures of the mandible and associated soft tissue injuries. It is believed that associated lacerations are to be thoroughly cleansed and meticulously sutured, and the underlying fractures cared for at a more optimum time unless open reduction is necessary.[4]

Even for the simple fractures treatment should be delayed until satisfactory roentgenograms of the bones of the face can be obtained. Complicated and serious fractures of the facial bones require a great deal of preoperative planning, and rather elaborate appliances may be needed for reduction and fixation.

Innumerable methods of reduction and immobilization have been described in the literature. Each has its advantages and disadvantages, and no one method is suitable for every case. It is our opinion that those procedures with which the surgeon is best acquainted and which he finds most satisfactory are the methods he should employ.

Attention can now be turned to a more detailed discussion of specific fractures.

*Nasal bones:* Fractures of the nasal bones usually do not present a great problem. Closed reduction and manipulation followed by immobilization with vaseline nasal packing and external splinting in most cases give satisfactory results. It is advisable to remove the splint, inspect the nose and reapply the splint at 48 to 72 hour intervals. However, when one is dealing with a severe crushing injury to the nose he must be aware that ethmoid fractures are not uncommonly associated with this type of injury. Consequently, every patient with a serious nasal injury should be examined for possible cerebrospinal rhinorrhea, and if this is present, reduction should be deferred for 10 to 14 days, and vigorous antibiotic therapy should be given during this period. Meningitis of the most serious nature is a constant threat, but usually following the interval of 10 to 14 days sufficient fibrous tissue has formed so that manipulation will not introduce infection to the meninges.

The more severely crushed nose can be reduced and fixed by elevation of the nose out of the face. Steel wire transfixion sutures over lead plates form an artificial buttress upon which the nose may rest. Surprisingly good results may be obtained by this relatively simple method of fixation even in the most severe injuries. Nasal bones unite very rapidly, but in spite of fibrous union they can be moved out of position with relative ease for approximately two weeks after the fracture. Therefore, splinting is advisable for a minimum of two weeks and sometimes longer.[1]

*Middle third of the face:* The types of fractures that one may encounter in this region are so many and varied that it is impossible here to discuss the treatment

of all of them in detail. Several excellent monographs have been written on the subject of middle third fractures, and the surgeon called upon to handle injuries in this area should be familiar with these.[2, 3, 5]

Because of its size, location, and attachments, a fracture of the malar bone itself is not a common injury. However, it is readily fractured at its attachments to the other bones and through the zygomatic arch, thus giving rise to a variety of deformities. When this solid bone is out of position serious ocular complications can and do develop. The most common of these is diplopia caused by a shift in position of the eyeball on the injured side. Also, fracture lines with few exceptions extend into the antrum, thus converting the fracture into an internal compound fracture with subsequent chance of infection and prolonged osteomyelitis. Because of the fact that the antrum is almost always involved in middle third fractures, the Caldwell-Luc approach through the superior labial sulcus is preferred for reduction of these fractures and adequate drainage of the sinus itself. Loose bone chips and blood clots commonly are seen and adequate evacuation of the sinus is necessary for proper treatment. In the study of this type of fracture adequate roentgenograms, made in the stereoscopic Waters position, are a necessity. Cloudiness of the antrum seen in these films is to us an indication for open antral reduction. In rare instances where such an approach is not necessary, external manipulation by means of a small hook or towel clip may be satisfactory. In some cases one may use the temporal approach of Gillies or any desirable combination. The Gillies approach is particularly indicated when one is dealing with a depressed fracture of the zygomatic arch which commonly impinges on the coronoid process of the mandible. When this occurs, the mouth can be opened only with difficulty, if at all.

Complete maxillary fractures allowing the middle third of the face to become elongated and freely movable are relatively common injuries. In this type the use of dental appliances is an aid. In general, we find little application for the use of the plaster head cap for the treatment of maxillofacial fractures. This is not a condemnation of the procedure, but satisfactory results can be obtained in most instances without its use. When there is malocclusion of the teeth due to maxillary fractures, the application of dental arch bars, followed by intermaxillary rubber band traction, produces results so dramatic as to astonish those not familiar with these injuries. This procedure, in conjunction with an elastic bandage applied around the head in the vertex-mandibular plane, or a chin strap attached to an ordinary operating room cap by means of rubber band traction with teeth in occlusion, will push the lowered maxilla into normal position where it becomes fixed in a remarkably short time.

Internal wire pin fixation for middle third fractures has a definite place, as does direct open reduction with wiring of fragments.[1] In general, however, we usually do not depend on internal wire pin fixation alone, but rather combine it with an antral approach and packing for postoperative fixation. These wire pins usually can be removed in two to six weeks according to the site of the fracture and degree of stability present.

*Mandible:* The management of mandibular fractures varies considerably with

the state of the teeth of the patient. If teeth of adequate quality, sufficient number and proper location are present, intermaxillary rubber band fixation is the treatment of choice in mandibular fractions. Tooth roots in the fracture line usually should be extracted unless the fracture occurs in the posterior molar region where the tooth root of the third molar actually may be holding the fragments in good position. In these days of antibiotic therapy the incidence of osteomyelitis is quite low, and the sweeping statement that routine extractions be done, now no longer holds true. Even the more severe mandibular fractures may be reduced by arch bars and intermaxillary traction when sufficient teeth are present.

The problem of fractures in the edentulous mandible is considerably different. The use of dental splints is a great help in many fractures which otherwise may have to be reduced and immobilized by the direct approach. Many may be fixed with splints and circumferential wiring. This is a most simple and satisfactory method when applicable. The use of Kirschner wires through the fragments is an acceptable and relatively simple method of fixation. The incidence of infection by this method is higher, however, than that occurring when direct visualization of the fragments and wire fixation is used. In most cases direct wiring is preferred. The use of external pin fixation is only rarely necessary. Such devices too frequently are used and invite complications. Considerable ingenuity is necessary in the care of many of these fractures, and the help of dental colleagues is to be encouraged.

## CASE REPORTS

Three cases illustrating many of the principles discussed above will now be presented.

*Case 1.* This 67 year old white man was admitted to the hospital approximately one hour after an automobile accident, a two car wreck in which his wife was killed. He suffered a scalp laceration, nose bleed, considerable pain about the lower jaw, and mental confusion. On being admitted to the neurosurgical service, he seemed alert, but was disoriented as to time and place. He had amnesia for the accident.

The general physical examination was entirely within normal limits with no evidence of long bone fractures or concealed abdominal injuries. There were multiple lacerations of the scalp, considerable swelling of the face, and bilateral ecchymosis about the eyes. The nose obviously was fractured, and there was considerable pain on motion of the jaw. On the night of the accident closure of the scalp lacerations was done under local anesthesia, and antibiotic therapy was instituted. The following day roentgenograms of the head revealed a fracture of the right parietal bone, a bilateral fracture of the mandible in the anterior portion of the body on one side and the midportion of the body on the other, with comminution of the fragments on one side (fig. 1). Stereoscopic Waters films showed bilateral fractures of the maxilla with comminuted fractures of both nasal bones. The patient was edentulous.

Nine days after the accident a bilateral open reduction with direct wiring of the mandible was done. The nasal fractures were manipulated and held in place with vaseline nasal packing and an external splint. The bilateral maxillary fractures were reduced readily by pushing the maxillas upward into place where they impacted into position and could be dislodged only with considerable force. It was believed then that when the patient reacted from anesthesia this fracture could be maintained with a head bandage to push the mandible against the maxilla. This procedure was done.

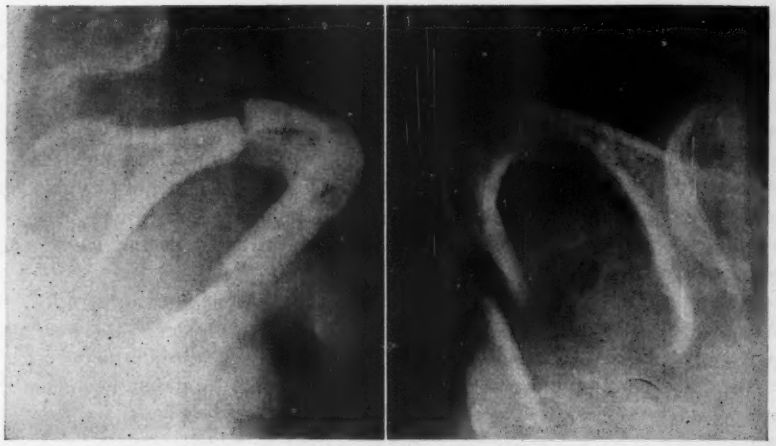

FIG. 1. Case 1. Oblique views of the mandible show clearly the bilateral fractures with comminution of the fragments on one side.

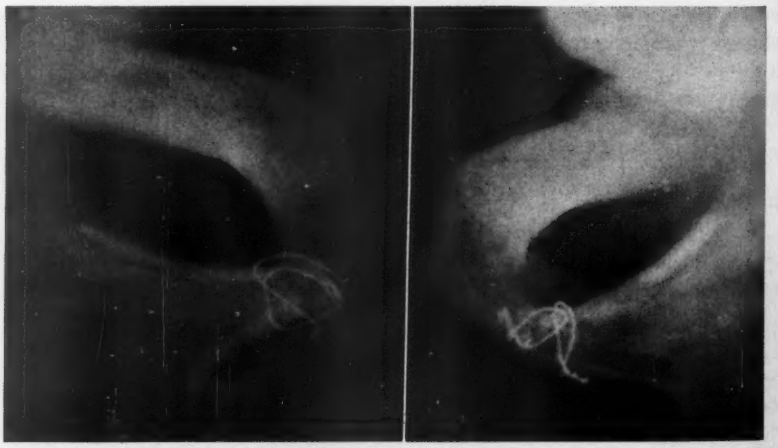

FIG. 2. Case 1. Roentgenograms six weeks following reduction and fixation show fragments to be in excellent position.

His postoperative course was entirely uneventful, and by the fifth postoperative day the maxilla was held in place firmly. Postoperative roentgenograms one month after operation revealed all bony fragments to be in excellent position and alignment (fig. 2).

*Case 2.* This 28 year old Negro man was admitted to the hospital two hours after he had been in an automobile accident. He received extensive lacerations of the lower lip with a fracture of the mandible. At the time of admission examination showed a massive laceration through the entire lower lip into the mouth, and across the chin, down to multiple fracture lines of the mandible (fig. 3). There was considerable respiratory difficulty. The patient was in mild shock with a blood pressure of 90/50. The pulse was weak and thready.

The remainder of the general physical examination was entirely within normal limits with no evidence of brain or internal fractures. Roentgenograms of the mandible showed

FIG. 3. Case 2. Preoperative photographs showing severe lip laceration with underlying fractures of the mandible.

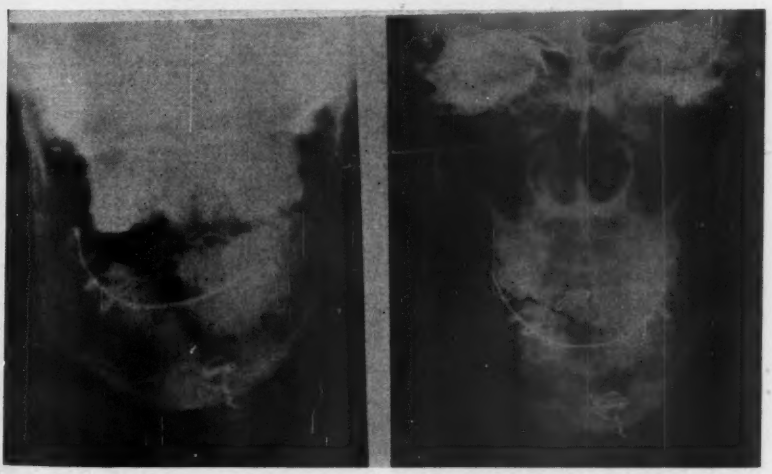

FIG. 4. Case 2. The roentgenogram on the left shows the position of the mandibular fractures both at the symphysis and at the right angle after direct fixation of the former, and following application of a lower arch bar. On the right is a roentgenogram made 48 hours later showing excellent reduction and the teeth in good occlusion.

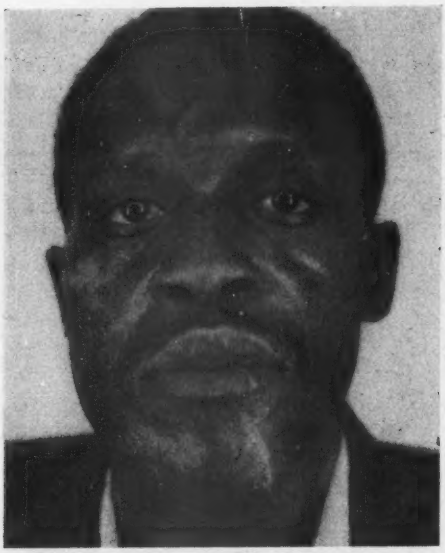

FIG. 5. Case 2. Postoperative photograph shows the final result approximately six weeks following the injury. There is some thickening in the scar.

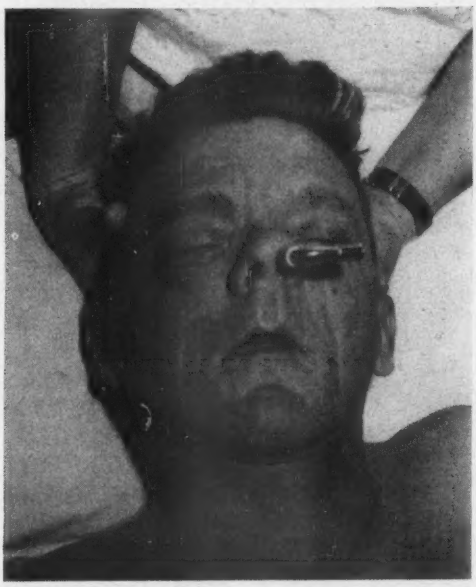

FIG. 6. Case 3. Preoperative photograph of the patient on the operating table. The increase in width of the right side of the face and the slough of the right lower eyelid and cheek can be seen.

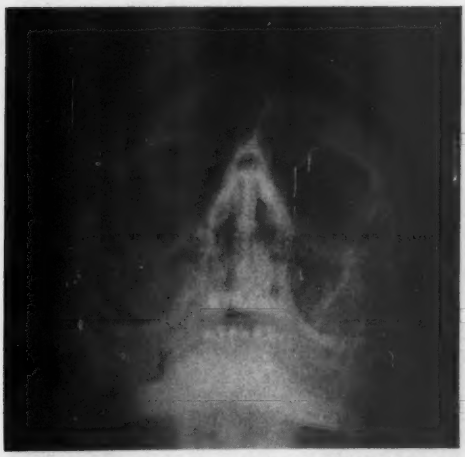

FIG. 7. Case 3. Preoperative roentgenogram shows clearly the outward and downward displacement of the zygoma from its frontal attachment with increase in the diagonal diameter of the orbit.

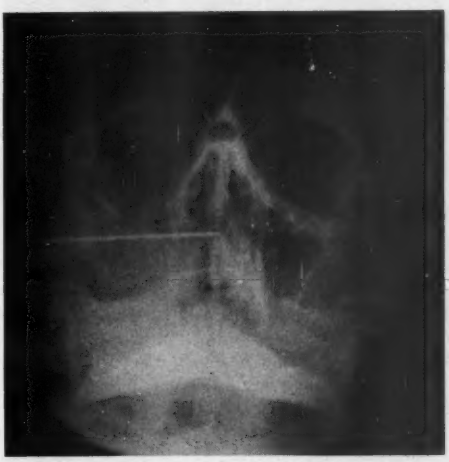

FIG. 8. Case 3. Roentgenogram following reduction shows good position and alignment of the facial fractures. The defect in the floor of the orbit is clearly seen. This defect was the result of bone loss at the time of the original accident. The bone fragment had been removed and thrown away.

a complete fracture through the horizontal ramus of the right mandible extending between the molar teeth. There was also a comminuted fracture of the mandible in the region of the symphysis.

The patient was given a clear airway, and blood transfusions were started. As soon as he was out of shock, he was taken to the operating room where the laceration was thoroughly cleansed and irrigated. Direct wiring was decided upon for fixation of the comminuted fracture of the symphysis. Lacerations were closed in the usual way.[4] Following this a buccal

FIG. 9. Case 3. Postoperative photograph approximately six weeks following operation. There is some edema of the lower eyelid and in the pedicle flap which was rotated into the area of slough overlying the bone. This has subsided considerably, but a minor operation on the lid would improve his result.

bar was placed about the mandibular teeth and the maxillary arch was wired for multiple loop traction. Traction was not applied at this time, however, due to the likelihood of vomiting following anesthesia. A tracheostomy was done at the close of the surgical procedure because of extensive lacerations of the floor of the mouth and associated edema. Thirty-six hours after operation rubber band intermaxillary traction was instituted, and the fragments were readily pulled into good position. His course was entirely uneventful, and six weeks after injury the wiring was removed from the teeth (fig. 4). Occlusion was excellent, and the condition of the facial scar was satisfactory (fig. 5).

*Case 3.* This 45 year old man was admitted to the hospital one week after he had been smashed in the face by a richocheting piece of timber 40 feet long by 1½ feet in diameter. This struck him in the right side of the face and caused very severe lacerations of the eyelids and cheek (fig. 6). He was knocked unconscious and obviously had facial fractures. The lacerations were repaired by his referring physician, who took care of him for several days until his state of consciousness improved so that he could be transferred.

On examination he had obvious fractures of the right side of the face with bulging in the region of the right zygoma and zygomatic arch. There was a soft tissue slough in the region of the outer canthus and over the right lower eyelid which exposed a fracture of the infraorbital ridge. Roentgenographic examination showed compound comminuted fractures of the maxilla, zygoma, zygomatic arch and comminution of the right coronoid process within the mass of the temporal muscle. There was a simple incomplete fracture of the right condyle in good position. There was also a fracture extending from the sigmoid notch to the angle. Most of the mandibular fractures were within muscle attachments and in good position. The zygoma was displaced outward a great distance and downward from its attachment with the frontal bone (fig. 7).

It was obvious that open reduction would be necessary. Accordingly, seven days after

his original accident he was taken to the operating room where some of his old incisions were reopened. The zygoma was approached through the antrum and elevated without great difficulty into its normal position with the frontal bone where it was fixed with stainless steel wires. This alone did not give adequate fixation since some bone had been lost from the infraorbital ridge and had been thrown away at his previous operation (fig. 8). A Kirschner wire was inserted—anchoring the bone in normal position. A pedicle graft was outlined and rotated on the right cheek to cover the area of slough of the eyelid. Postoperatively he did well. The Kirschner wire was removed five weeks following operation after postoperative roentgenograms showed all bones in good position, and clinical examination showed adequate fixation of the fragments.

At the present time he has very poor vision in his right eye due to internal derangement at the time of the injury. He also has some scarring of the lower eyelid which readily could be repaired by a plastic procedure. However, the patient does not want this done for he is satisfied with the appearance of his face and the function of his jaw (fig. 9).

## SUMMARY

The general aspects of maxillofacial injuries are discussed.

A plan of treatment for the more common injuries of the facial bones is presented. It is not intended to state that these methods are the only satisfactory ones, but ones which have proved to give good function and appearance.

Cases are presented illustrating various types of fractures and their management.

## REFERENCES

1. Brown, J. B., Fryer, M. P., and McDowell, F.: Internal wire-pin fixation for fractures of upper jaw, orbit, zygoma and severe facial crushes, Plast. & Reconstruct. Surg. *9:* 276 (March) 1952.
2. Erich, John B., and Austin, Louie T.: Traumatic Injuries of Facial Bones: An Atlas of Treatment. In Collaboration with Bureau of Medicine and Surgery, U. S. Navy, Philadelphia and London, W. B. Saunders Company, 1944.
3. Fry, W. Kelsey, and others: The Dental Treatment of Maxillo-Facial Injuries, New York; Wm. Salloch; Oxford: Blackwell, 1942.
4. Lyons, C., and Upchurch, S. E.: Symposium on minor surgery: management of common superficial wounds, S. Clin. North America (*31:* 1271 (Oct.) 1951.
5. National Research Council, Division of Medical Sciences, Manual of Standard Practice of Plastic and Maxillofacial Surgery. Prepared and edited by the Subcommittee on Plastic and Maxillofacial Surgery of the Committee on Surgery of the Division of Medical Sciences of the National Research Council, and Representatives of the Medical Department, U. S. Army. Robert H. Ivy, Chairman. (Military Surgical Manuals, Volume I.) Philadelphia and London, W. B. Saunders Company, 1942.

# POSTCHOLECYSTECTOMY AMPUTATION NEUROMA*

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The unexpected recurrence of symptoms following cholecystectomy for cholecystic disease is not common; when it occurs it may present a difficult diagnostic problem. It has been the general opinion that, following the removal of the gallbladder in those people who give a history of biliary colic, nausea, vomiting, and indigestion, and in whom there has been a demonstration of biliary calculi by cholecystography, one may expect 80 to 95 per cent to be relieved of their symptoms. In the absence of biliary colic and the demonstration of a nonfunctioning gallbladder without included calculi by the Graham-Cole Test the removal of the gallbladder is said to produce no clinical improvement in 40 per cent of the patients.[6] However, failure to demonstrate the gallbladder in those patients who have had colic usually is excellent evidence of the presence of noncalcareous stones. It is in those patients, in whom the expectation for the relief of symptoms is great, that the unexpected recurrence of pain following cholecystectomy is so distressing. This rather sizable group of patients often is referred to as suffering from the postcholecystectomy syndrome.

One of the most common causes of the recurrence of symptoms undoubtedly is the presence—or persistence—of a stone or stones within the common or hepatic bile duct. The reasons for this are various. Best[2] has shown that 7 per cent of patients with cholelithiases also have hepatic stones. These hepatic stones likely will be overlooked. Hicken[8] has suggested that 95 per cent of secondary operations upon the common bile duct are for stones left from the previous operations.

Hypertonicity or spasticity of the ampulla of Vater has been proposed as an explanation for the reflux of bile into the pancreatic ducts. The resulting irritation may produce symptoms which closely resemble biliary disease. Since there is a common passage between bile duct and pancreatic duct in only 20 per cent of people it is not surprising that reflux of bile is an unusual complication.

There still are other causes of the symptoms associated with the postcholecystectomy syndrome. Recently, it has been demonstrated that fibrosis of the sphincter of Oddi[3] may be responsible for the recurrence of symptoms in some patients. The common bile duct or the hepatic duct may be injured at the original operation. A malignant tumor may be overlooked at the time of cholecystectomy which, of course, will continue to produce symptoms during the postoperative period. In still other patients, the persistence of a low-grade infection of the biliary system results in the continuation of symptoms.[13] Improvement in such individuals is always slow and questionable. It has been the opinion of Gray and Sharpe,[7] and also of Garlock and Hurwitt,[5] that the persistence of

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the cystic duct stump in some patients results in the recurrence of symptoms. They are of the opinion that removal of this stump results in improvement in the majority of individuals. This is a possible, but not a common cause of symptoms.

In 1940 Benson[1] reported results obtained by necropsy examinations of patients who previously had had cholecystectomies. In those patients who had complained of distress in the years following operation 14 per cent showed normal appearing bile ducts while 86 per cent had bile ducts which were much dilated. In contrast he observed that the bile ducts were normal in size in 83 per cent of those patients who had had no postoperative biliary symptoms.

The gallbladder and the biliary tree are abundantly supplied with nerves. It seems logical to anticipate that, considering the large numbers of operations done in this area, abnormalities in function and regeneration of these nerves may occur. The nerves[10] about the bile duct area have both sympathetic and parasympathetic derivations. Starting at the celiac plexus and extending along the biliary tree, an anterior and posterior hepatic plexus with many communications between may be identified. The anterior plexus always follows the hepatic artery. The posterior plexus passes to the right from the right celiac ganglion and lies superiorly in the portal area between the portal vein and the common bile duct. Hinton[11] has shown that fibers from the anterior hepatic plexus pass along the gastroduodenal artery and that fibers from the gastroduodenal plexus reach the terminal portion of the common bile duct. From the medial aspect of the retroduodenal portion of the bile duct he has traced fibers of the posterior hepatic plexus to the ampullary portion of the bile duct. The area of maximum concentration of nerve fibers, however, is in the triangular region formed by the cystic and hepatic ducts. From this region lateral and medial nerves have been observed passing to the gallbladder.

Womack, in 1938, reported changes in the tissues surrounding the gallbladder following an episode of inflammation.[14] He noted that following a severe attack of cholecystitis, there were definite pathologic changes in and around the nerve fibers of the gallbladder with the result that the nerves were surrounded by poorly vascularized fibrous tissue. The ensuing ischemia and associated changing tensions on the nerves so altered their threshold of stimulation that stimuli, which normally had not been apparent to the individual, were now of significance. Following the removal of the gallbladder and its associated lesions there was much improvement in the patients. It was his opinion that at the point where the cystic duct was ligated and transected there was not only inflammation with its resultant fibrous tissue proliferation but also nerve trunk regeneration which occasionally resulted in the production of an amputation neuroma. This result also could occur at any point along the biliary tree where these nerves were traumatized either at operation or by infection.

In 1946 Cieslak and Stout[4] reported the first recorded case of a neuroma occurring after a cholecystectomy. This neuroma had produced symptoms of a severe nature. A second operation was done at which time the neuroma was found and removed. In 1947 Womack and Crider[15] reported 6 more cases with similar find-

ings. Each of these patients had had a cholecystectomy followed by a recurrence of symptoms. These were so severe that a second operation was advised. In 4 patients the cystic duct stump was identified. It was hard and fibrous and the site of an amputation neuroma. This was confirmed in each instance by microscopic examination. In the remaining 2 patients neuromas in the periductal tissues were demonstrated.

In our clinic during the past eight years, 9 patients have been seen who have had postcholecystectomy amputation neuromas. Each patient, after a period ranging from 2 to 13 years after cholecystectomy, was reoperated upon because of symptoms such as have been described. At the second operation, a remnant of the cystic duct stump was found in 7 patients. The cystic duct stump was very hard and fibrous. It was believed at the time of operation that this could represent a neuroma and such impressions were confirmed by microscopic examination of the tissue removed. In the remaining 2 patients an obstruction of the bile duct near the point of entry of the cystic duct was observed. Removal of the obstruction and microscopic examination of this tissue showed that it represented an amputation neuroma.

## CASE REPORTS

*Case 1.* S. S., a woman, aged 51, first noted episodes of right upper quadrant pain radiating to the back in 1925. A cholecystectomy was done in 1933. She was improved for 16 years following the operation. In August 1949, and continuing until her admission in December 1950, she had intermittent episodes of right upper quadrant pain associated with nausea and vomiting. Occasionally following an attack, light colored stools and dark urine were observed. At the time of admission to the University Hospital she was having attacks of pain daily. Laboratory and roentgen examinations prior to her second operation were not suggestive of any specific disease. At operation in December 1950, a slightly enlarged common duct was observed. Adjacent to the cystic duct remnant a mass of fibrotic tissue was noted and removed. The pathologist reported, "Cystic duct with a portion of an amputation neuroma in the wall" (fig. 1). At the time of the patient's last examination in July, 1952, she was completely asymptomatic.

*Case 2.* M. B., a woman, aged 38, on the basis of a previous history of biliary colic, had a cholecystectomy at another hospital in 1944. Starting one month after her operation and continuing until her admission to the University Hospital in July 1951, this patient had multiple episodes of right upper quadrant pain, nausea, and vomiting. The pain was relieved only by the use of hypodermic injections of a narcotic. Following her admission to the University Hospital in July 1950, extensive laboratory and radiographic studies were done. All of these examinations were within normal limits. At the time of her operation, the common duct was noted to be of normal size and without stone. The cystic duct stump was scarred and fibrotic. The cystic duct stump and associated scar were removed. Pathologic examination showed this to be, "An amputation neuroma adjacent to tissue compatible with cystic duct." At the present time, 20 months after operation, she is free of all symptoms relating to her previous disease.

*Case 3.* H. B. was a woman 53 years of age. In February 1950, following a 15 year history of vague right upper quadrant pain, intolerance to fatty food and the demonstration of stones in the gallbladder, the patient had a cholecystectomy. One month following the operation, she had the onset of severe right upper quadrant pain necessitating the administration of narcotic for relief. At the time of entry to the University Hospital she required narcotics daily for comfort. At operation, 24 months following the onset of symptoms, the common bile duct was not enlarged and no stones were found within it. A cystic duct rem-

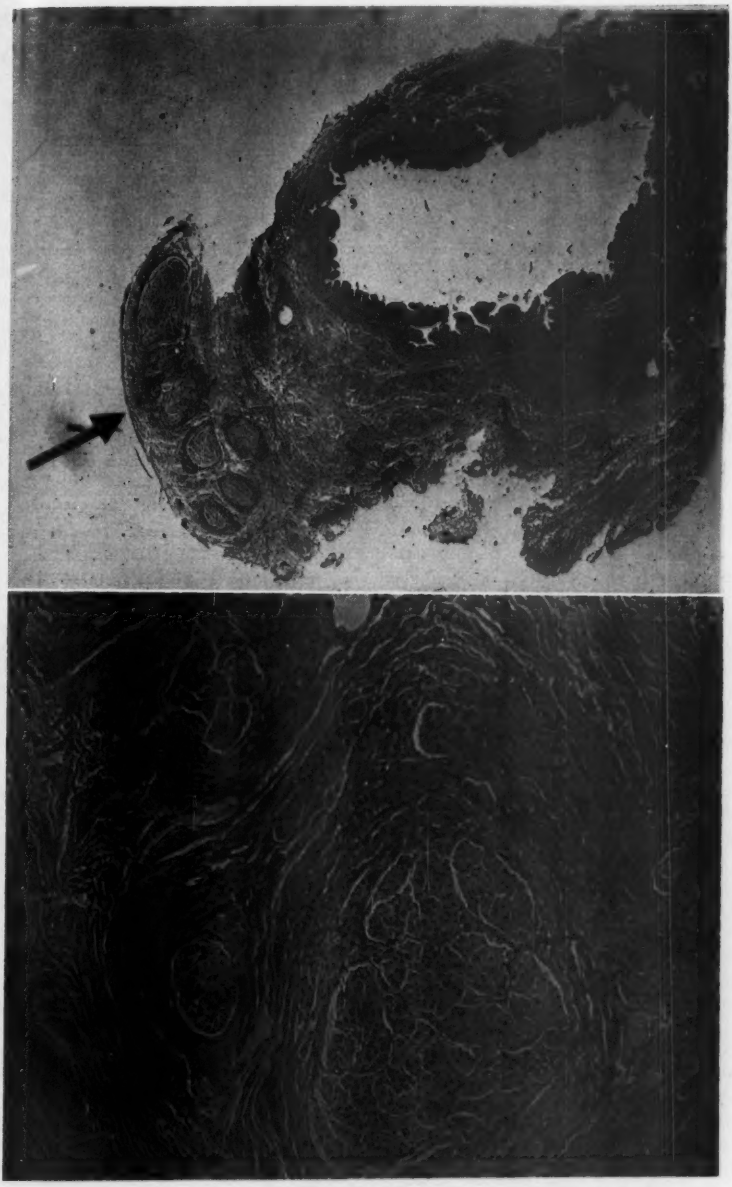

FIG. 1. Remnant of cystic duct with an amputation neuroma in its wall

nant was found and removed. It was the opinion of the surgeon that this well might be an amputation neuroma. This diagnosis was confirmed by the pathologist who reported, "An amputation neuroma in the stump of the cystic duct." Since her operation she has had no recurrence of her symptoms.

*Case 4.* G. R. was a woman aged 48. In 1938, following a history of jaundice and the demonstration of stones within the gallbladder, this patient had a cholecystectomy. Within one month of the operation she began having episodes suggesting severe biliary colic, with nausea, vomiting, chills, fever and slight jaundice. The pain was controlled only by the use of narcotics. Thirteen years following the onset of these symptoms she entered the University Hospital. Preoperative studies included many laboratory and radiographic studies. Each study was within the normal limit. At operation in March 1950, no stones were found in the common bile duct. A remnant of the cystic duct was observed and believed to be the site of an amputation neuroma. This was removed. The clinical impression was substantiated by the pathologist who reported, "An amputation neuroma in the wall of the cystic duct" (fig. 2). Three years following her operation she has had no recurrence of her symptoms.

*Case 5.* N. G. was a man, aged 60, who had his first right upper quadrant pain requiring narcotics in 1919. He did well following this episode until 1942 when he had the onset of recurrent attacks of biliary colic, chills, fever, and slight jaundice. In 1943 he had a cholecystectomy and and exploration of the common duct. He was asymptomatic for three years following which he had the recurrence of frequent attacks of pain, nausea, vomiting, chills, fever, and malaise. Laboratory and roentgen examinations prior to his operation were all normal. In December 1946, an exploration of the common duct was done. The common duct was moderately dilated but no stones were found. The cystic duct stump was identified and noted to be enlarged and an area of firmness within it suggested an amputation neuroma. The cystic duct stump and associated neuroma were excised. The clinical impression was substantiated by the pathologist who reported, "An amputation neuroma in the wall of the specimen which suggests a gallbladder." At the time of his last visit here, seven years following operation, he was without symptoms.

*Case 6.* V. R. was a woman, aged 49 and a patient of Dr. H. M. Bishop, Saginaw, Michigan. In July 1944, following a history of episodes of severe right upper quadrant pain not associated with nausea, vomiting or jaundice, a cholecystectomy was done. Two months following her discharge from the hospital she had the onset of recurrent episodes of severe right upper quadrant pain frequently necessitating the administration of a narcotic for relief. Preoperative laboratory examinations were within the normal limits. At the time of the second operation in 1948 a large cystic duct stump was found and removed. Examination of the tissue by the pathologist showed, "Many bundles of nerve fibers somewhat tortuously coiled in scar tissue. This qualifies as an amputation neuroma." At the present time, five years after her operation, she is symptom free.

*Case 7.* F. M. was a man, aged 51. For 10 years prior to his cholecystectomy and choledochotomy in April 1952, this man had had intermittent episodes of right upper quadrant pain. These were associated with nausea and intermittent jaundice. Starting two months after his operation and continuing until his admission in June 1953, he had episodes of severe right upper quadrant pain which required narcotic injections. Also noted was occasional nausea and vomiting and slight jaundice. At the time of his second operation no stones were found in the common duct. The cystic duct stump was very hard and fibrous. Histologically it was reported as "large nerve fibers included in fibrous connective tissue." At the present time he has had no recurrence of symptoms.

*Case 8.* P. J. was a woman, aged 47. In August 1951, following an 11 year history of episodes of midepigastric pain associated with light colored stools, dark urine and nausea, a cholecystectomy was done in another hospital. The patient's postoperative course was complicated by bile peritonitis, recurrent jaundice, and pneumonia. Prior to her entering the University Hospital in January 1952, she had had several episodes of epigastric pain associated with jaundice, clay-colored stools, and dark urine. She also had nausea, vomiting, chills and fever. At the time of her operation in January 1952, a stricture of the bile

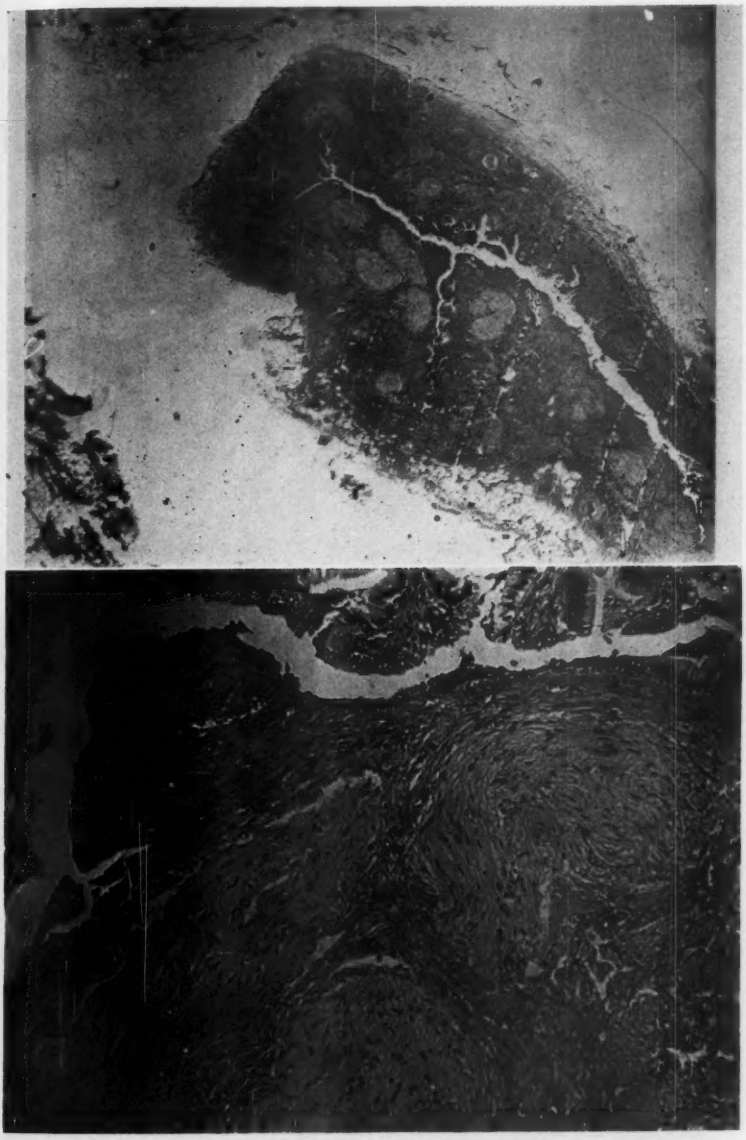

FIG. 2. High and low magnification of cystic duct remnant and neuroma

duct 3 cm. in length was found. This was excised and the continuity of the duct re-established. Microscopic examination of the tissue removed showed it to be composed of, "an amputation neuroma in connective tissue" (fig. 3). At the present time she is much improved. She still suffers from occasional episodes of chills and fever.

*Case 9.* M. O. was a man, aged 48. In May 1948, while having a physical and roentgeno-

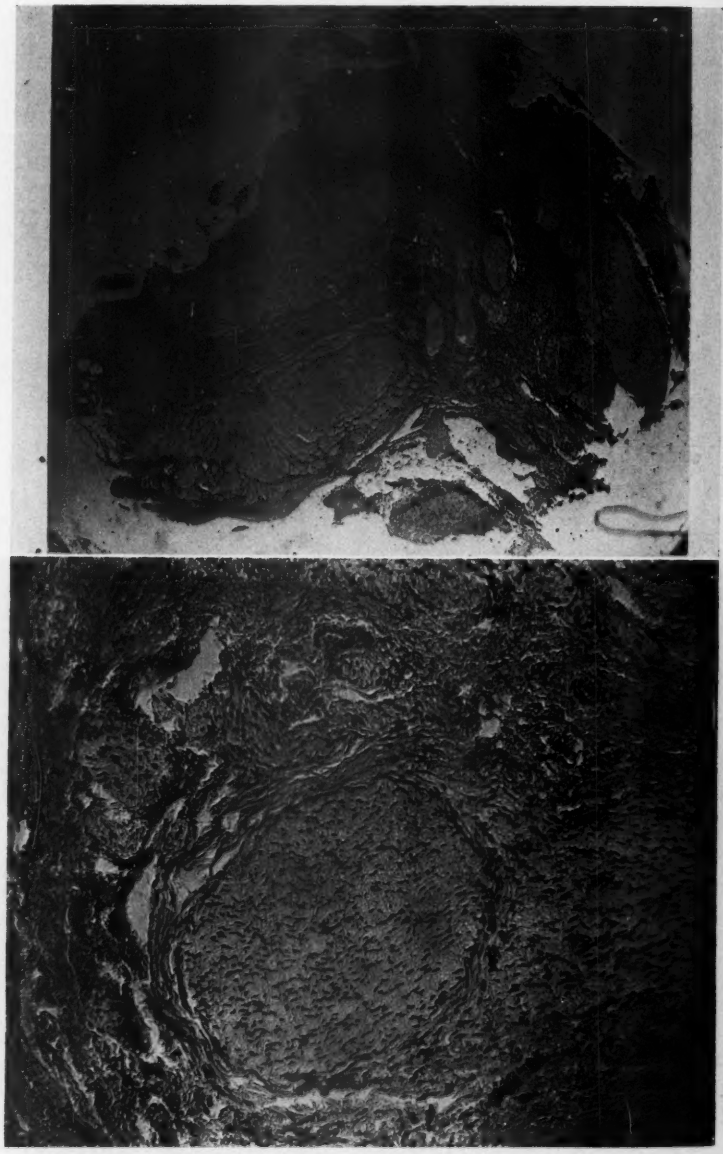

FIG. 3. High and low magnification of tissue from common duct region showing a neuroma

logic examination, cholelithiasis was observed. Cholecystectomy was done following which he developed a biliary fistula. In June 1949, he had an exploration of the common duct with anastomosis of the duct. Following this operation he was well until August 1950. He then noted pruritic jaundice, chills and fever. He was admitted to the University Hospital,

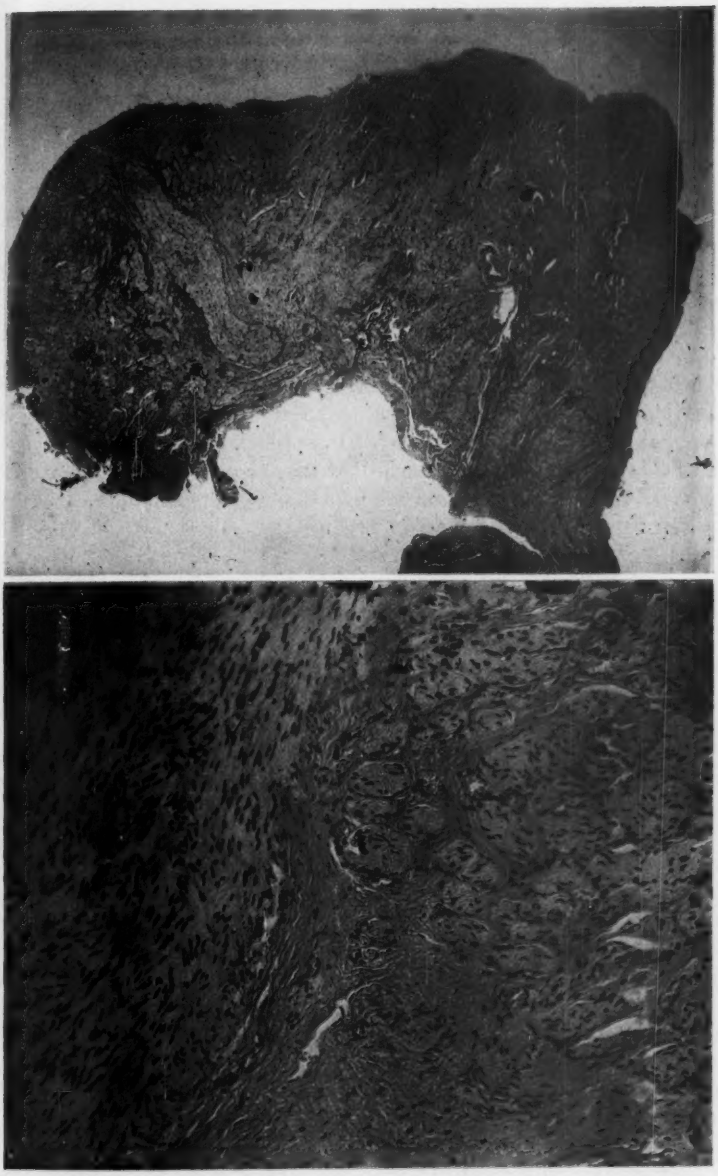

FIG. 4. Section of common bile duct region showing a neuroma in scar tissue

where on Aug. 14, 1950, a re-exploration of the common duct was done. An obstruction was found and excised and a re-anastomosis of the biliary passage was made over a T tube. Microscopic examination of the obstructed segment showed an "amputation neuroma in scar tissue" (fig. 4). Since the second operation the patient has been asymptomatic, and, at the present time, 31 months after operation, is in good health.

## DISCUSSION

A bare summary of symptoms and findings of the first 7 patients of this series are presented in chart I. For comparison, the patients of Womack (1947) and those of Troppoli and Cella (1953) are also recorded. The outstanding feature in all of these patients was right upper quadrant pain not easily distinguished from biliary colic. This pain usually was severe in nature and often demanded some form of narcotic for relief. While not as frequently observed, nausea and vomiting were also of common occurrence.

Prior to the suggestion of an operation, each patient in this series was carefully studied. Upper and lower gastrointestinal roentgen studies usually were done, and a number of the standard tests of liver function and bile excretion were reported. These tests are significant in that they showed no positive evidence of the source of the disease. Operation was therefore suggested on the basis of clinical impression rather than on objective findings.

In each of the 7 patients the surgeon, at the time of operation, was able to identify and expose the cystic duct remnant which was found in each patient to be enlarged. Clinically it appeared to be firm, scarred, and fibrotic. The common duct was explored in each patient and found dilated in 3. A small stone 0.5 cm. in diameter was found in one patient and removed (case #1).

After operation the 7 patients with cystic duct neuromas had immediate, complete relief of symptoms and at the time of their last examinations had experienced no recurrence of symptoms. These results are expected and are in accord with other reports. In general, the recurring episodes of pain which mark the presence of a neuroma have appeared within a few months of the operative trauma which induced them. The reappearance of symptoms as long as one or more years after operation is most unusual, and a diagnosis of amputation neuroma in such an instance should be looked upon with some skepticism. An example of this is seen in the first patient described above. Most surely a portion, perhaps all, of this patient's symptoms related to the presence of a calculus within the common bile duct. It seems unnecessary to emphasize that careful exploration of the common bile duct must be done at the time a neuroma is found and removed. Our ability to distinguish colic from the pain of a neuroma has been of fairly low order prior to operation and operation is commonly done on a diagnosis of calculus disease. The unexpected finding of an amputation neuroma must not deter the surgeon from completing his initial undertaking.

It seems desirable to re-emphasize this entity as a cause of pain in the patient with "severe colic without stones after cholecystectomy."[9]

Kjaergaard[9] recently has reported 12 cases of patients upon whom a choledochoduodenostomy was done when re-exploration, after cholecystectomy, failed to demonstrate a calculus within the duct. One patient, free of symptoms for two

CHART I

*Summary of symptoms and findings in the first 7 patients of our series. These are compared with those of Womack and Troppoli and Cella*

| | Univ. Hosp. Series (1953) | | | | | | | Womack (1947) | | | | | | Troppoli & Cella (1953) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 1 | 2 | 3 | 4 | 5 | 6 | 1 | 2 |
| Pain | X | X | X | X | X | X | X | X | X | X | X | X | X | X | X |
| Narcotic Needed | | X | X | X | X | X | X | | | | | | | | |
| Nausea | X | X | | X | X | | X | X | | X | X | | | | |
| Vomiting | X | X | | X | X | | X | X | | X | X | | | | |
| Jaundice | X | | | X* | | | X* | X | X | | | | | | |
| Chills | | | | X | X | | | | | | | | | | |
| Fever | | | | X | X | | | | X | | | | | | |
| Lab. Exam. Normal | X | X | X | X | X | X | X | X | X | | X | | | X | X |
| X-Ray Exam. Normal | X | X | X | X | X | | | X | | | X | | | | |
| Improved with op. | X | X | X | X | X | X | X | X | X | X | X | X | X | X | X |

* By history.

months, required a splanchnicectomy for relief of symptoms. Four patients only are reported as fully recovered. It seems quite possible that in some of these 12 patients with *severe postoperative dyskinesia* a more painstaking search might have revealed amputation neuromas.

Cases 8 and 9 illustrate a symptomatic amputation neuroma observed at other points in the biliary tree where there has been nerve injury and associated inflammation. The presenting symptoms in each were those of biliary obstruction. At the time of operation, a hard, firm obstruction was found in each. This was removed and the continuity of the biliary tree was re-established. In each instance the pathologist believed that the tissue represented an amputation neuroma.

## CONCLUSIONS

Injury of the nerves immediately adjacent to the cystic duct may result in the formation of an amputation neuroma. The surrounding inflammatory process may so alter the threshold of stimulation that symptoms are produced. The disease is characterized by episodes resembling biliary colic, usually associated with nausea and vomiting. Little help is gained by the use of the standard roentgen and blood examinations. Surgical removal of the cystic duct stump and its associated amputation neuroma has resulted in cure.

Neuromas may appear any place along the hepatic or common bile ducts in the nerves of the hepatic plexus. The occurrence of this complication can be minimized by careful sharp dissection of the sympathetic nerve fibers from the cystic duct before it is ligated.

## REFERENCES

1. Benson, K. W.: Dilatation of bile ducts and its relation to distress after cholecystectomy, Am. J. Digest. Dis. *7:* 1 (Jan.) 1940.
2. Best, R. R.: Incidence of liver stones associated with cholelithiasis and its clinical significance, Surg., Gynec. & Obst. *78:* 425 (April) 1944.
3. Cattell, R. B., and Colcock, B. P.: Fibroisis of spincter of Oddi, Ann. Surg. *137:* 6, 797 (June) 1953.
4. Cieslak, A. K., and Stout, A. P.: Traumatic and amputation neuromas, Arch. Surg. *53:* 646 (Dec.) 1946.
5. Garlock, J. H., and Hurwitt, E. S.: Cystic duct stump syndrome, Surgery *29:* 833 (June) 1951.
6. Graham, E. A., and Mackey, W. A.: Consideration of stoneless gallbladder, J.A.M.A. *103:* 1497 (Nov. 17) 1934.
7. Gray, H. K., and Sharpe, W. A.: Biliary dyskinesia; role played by remnant of cystic duct, Arch. Surg. *46:* 564 (April) 1943.
8. Hicken, N. F., Stevenson, V. S., Franz, B. J., and Crowder, E.: Technic of operative cholangiography, Am. J. Surg. *78:* 347 (Sept.) 1949.
9. Kjaergaard, S.: Choledochoduodenostomy in postoperative dyskinesia, Acta chir. scand. *104:* 2–3, 87 (Dec. 10) 1953.
10. Raigorodosky, J. L.: Die Nerven der Leberpforte des Menschen, Ztschr. f. d. ges. Anat. (Abt. 1) *86:* 698 (July 31) 1928.
11. Shafiroff, B. G. P., and Hinton, J. W.: Surgical anatomy of choledochal nerves, Arch. Surg. *60:* 944 (May) 1950.
12. Troppoli, D. V., and Cella, L. J., Jr.: Postcholecystectomy syndrome, Ann. Surg. *137:* 2, 250 (Feb.) 1953.
13. Wier, J. F., and Snell, A. M.: Symptoms that persist after cholecystectomy; their nature and probable significance, J.A.M.A. *105:* 1093 (Oct. 5) 1935.
14. Womack, N. A.: Pathologic changes in chronic cholecystitis and production of symptoms, Surgery *4:* 847 (Dec.) 1938.
15. Womack, N. A., and Crider, R. L.: Persistence of symptoms following cholecystectomy, Ann. Surg. *126:* 31 (July) 1947.

# CONGENITAL ANOMALIES OF THE GALLBLADDER

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Malformations of the bile ducts and their associated blood vessels have been exhaustively studied and described by Eisendrath,[10] Holmes,[14] Kehr,[16] Howard and Walbach,[15] Mentzer,[20] Ladd,[18] and others. The surgical significance of these anomalies is constantly being emphasized and rightly so, for they are frequently encountered and failure to recognize them may lead to surgical disaster.

Congenital anomalies of the gallbladder are much less frequently encountered, but it is still necessary for the surgeon and the roentgenologist to familiarize themselves with the malformations of this organ which is so often the subject of roentgenologic examination and surgical treatment.

In 1926 Boyden[3] made an anatomic and embryologic study of this subject in 9,221 cadavers and his classification has served as a guide since that time. In 1936 Gross[11] reviewed the literature on anomalies of the gallbladder and presented a study of their clinical aspects. His review of 148 reported cases, with a case of his own, has been a valuable reference for subsequent writers. The information contained in the papers of Boyden and Gross has been freely used in this article (fig. 1).

We will discuss the topic under three headings: anomalies of form, anomalies of position, and absence of the gallbladder.

## ANOMALIES OF FORM

*Double gallbladder:* This term refers to duplications of the viscus in which there are two separate gallbladder cavities and two separate cystic ducts. The two ducts may converge and form one cystic duct before they drain into the common duct (Y shaped type) or they may drain separately into the ductal system. Where they drain separately they may both empty into the common duct or one may empty into one of the hepatic ducts. In a rare type, which will be described later, the cystic duct of the accessory viscus plunges directly into the liver substance.

Both vesicles may be approximately the same size or the accessory gallbladder may be considerably smaller as was the case in both of our specimens removed at operation. In the case reported by Gross[11] the accessory gallbladder was considerably larger than the normal one. The two structures may lie in apposition in the gallbladder fossa and be covered by a common serosal coat, thus obscuring their duplicate nature. This finding was present in both of our surgical specimens. More often the double nature of the organs is made evident by a fissure between them. The structures may be separated with the normal gallbladder lying in its fossa and the accessory structure located elsewhere. Some of

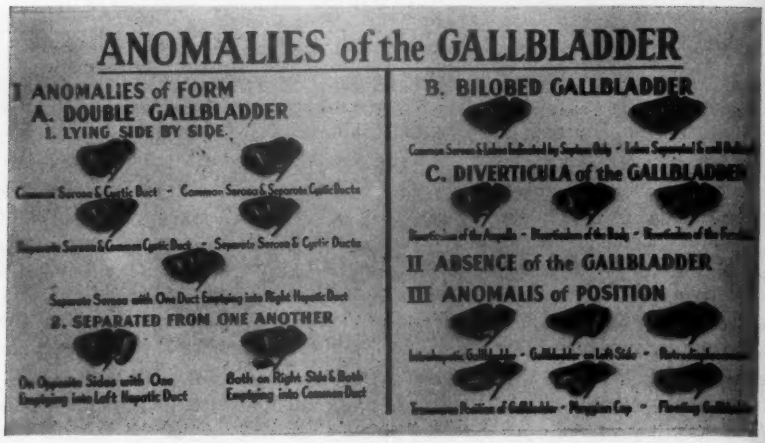


FIG. 1. Illustrations of the three types of anomalies of the gallbladder

the reported positions have been under the left lobe of the liver, embedded in the right lobe of the liver and within the gastrohepatic ligament.

The embryologic explanation of the formation of these double structures varies with the form they take. An accessory gallbladder which connects directly with the cystic, common or one of the hepatic ducts, is believed to arise from one of the small cellular outpocketings frequently seen on these structures in embryonic life. These rudimentary duct buds are common in human embryos and may spring from any one of the main ducts. However, they are most numerous at the junction of the two hepatic ducts with the common duct.[17] These buds normally regress as the fetus develops and it is believed that the persistence and growth of one of them results in the formation of a supernumerary gallbladder. If the bud from which the accessory gallbladder develops originates on one of the hepatic ducts or the common duct, the supernumerary vesicle will have a complete cystic duct of its own. If the anlage in question originates from the cystic duct, the accessory gallbladder and the normal viscus will be connected with the common duct by a Y shaped cystic duct.

The other embryologic type of supernumerary gallbladder is that which has been designated trabecular gallbladder by Boyden, because it is believed to arise from the hepatic trabeculae near the gallbladder fossa. This type of viscus lies beside the normal one, but instead of emptying into one of the main ductal structures its cystic duct plunges directly into the liver substance. It is believed to arise from an outpouching of the liver cords and it connects with the smaller bile capillaries. The only known case of a trabecular gallbladder in man was reported by Croudace[8] in 1931.

None of the reports in the literature give any indication of a characteristic symptom or symptom complex which might enable one to diagnose this congenital anomaly clinically. When a double gallbladder is the site of inflammation or

stone formation, the signs and symptoms are indistinguishable from those found in the usual case of cholecystitis or cholelithiasis.

The fact that most of these cases have been found at operation and not at autopsy would lead one to believe that this anomaly is more prone to inflammation and stone formation than the normal gallbladder. This probably results from the more frequent occurrence of imperfect biliary drainage due to the anomalous arrangement of the cystic ducts.

If a diagnosis of double gallbladder is made preoperatively, it has to be made on roentgenologic evidence. The first case to be demonstrated by cholecystography was reported by Climan[6] in 1929. Usually two distinct gallbladder shadows can be visualized. Films taken after the administration of the fatty meal may reveal that one viscus empties faster than the other and makes the diagnosis more definite. Nichols[22] and Sherren[23] both reported cases in which the roentgenologic diagnosis was made by visualizing distinct calculi within the organ. Verstandig and Moore[26] reviewed the literature in 1948 and found 12 cases of double gallbladder which had been diagnosed roentgenologically. They added 1 case of their own, bringing the total up to 13.

We have been able to find reports of 45 cases of double gallbladder in the literature. We present 4 additional cases. Two of these were proved at operation, the other 2 were not confirmed surgically.

*Case 1.* H. W. B., a 32 year old, white, married woman had right upper quadrant soreness and moderate intolerance to fatty foods for five years before admission to the hospital. For two years before admission she had had repeated attacks of gallbladder colic. These attacks were usually associated with nausea and vomiting and lasted from two to four days. There was no history of jaundice. Since the onset of gallbladder colic there had been intolerance to fatty food.

Her past and personal histories were noncontributory and the system review did not reveal any pertinent information.

Admission physical examination revealed moderate tenderness to deep palpation in the right upper abdominal quadrant. A cholecystogram showed two shadows, one being larger than the other. The larger shadow contained a small nonopaque calculus. The smaller contained several opaque stones (fig. 2).

At operation it appeared that the roentgenologic diagnosis was incorrect. The gallbladder seemed to be a large, tense viscus with thickened walls containing several palpable stones. The cystic duct was delineated and clamped flush with the common duct. When this structure was divided, two lumens were visible and further investigation revealed that these comprised a double cystic duct, the two lumens of which emptied into the common duct in juxtaposition to each other. These two cystic ducts were covered by a single serosal coat. What appeared to be a single gallbladder was removed. No other intra-abdominal anomaly was found.

When the surgical specimen was dissected after the operation, it was found to consist of two complete gallbladders covered by a single serosal coat (fig. 3).

The smaller of the specimens was filled with a greenish gelatinous material. Its walls were thick and contained several moderate sized, smooth, yellow, slightly granular stones. On microscopic examination the walls of this viscus showed scarring and lymphocytic infiltration throughout the entire thickness. These stones on analysis proved to be combination calculi with bilirubin centers and cholesterol exteriors.

The second gallbladder was much larger than the first and its wall was not as thick. It contained a thin, watery bile, quite different from the gelatinous material described

FIG. 2. Case 1. Cholecystogram showing two distinct gallbladders

above. The mucosa of this viscus was stippled throughout with fine yellow projections. This bladder also contained several stones which were larger than the ones described above and much darker. On microscopic examination the walls of this gallbladder showed slight scarring. The mucosa was thrown into folds. The mucosa on the crests of many of these folds contained pseudoxanthoma cells. The stones in this viscus, on analysis, proved to be bilirubin calculi.

This patient's postoperative course was uneventful. Since discharge she has been taking an unrestricted diet without difficulty.

*Case 2.* This patient, a 60 year old man, had been having attacks of right upper quadrant pain without radiation for 40 years. These attacks were occasionally accompanied by nausea

FIG. 3. Case 1. Roentgenogram of specimen removed at operation

and vomiting and had been becoming more frequent and more severe before admission to the hospital. There had never been any noticeable jaundice.

Cholecystograms taken nine years before admission did not show any visualization of the gallbladder. On admission physical examination there was tenderness without spasm over the right upper quadrant of the abdomen. Cholecystograms, after a double dose of dye, again did not visualize the gallbladder.

A diagnosis of chronic cholecystitis was made. At operation what appeared to be an enlarged gallbladder with a pouch in the region of the ampulla, extending down over the common duct, was found. The gallbladder did not contain any stones and the common duct was normal. The gallbladder and appendix were removed. The common duct was not explored.

When the removed viscus was examined postoperatively, it was found to consist of two gallbladders covered by a common serosa. One was much smaller than the other and this one had formed the pouch-like projection described above, which had extended down over the common duct. Each viscus had a short cystic duct and these united to form a short common segment which emptied into the common duct in the usual manner.

Microscopic examination of the larger bladder showed lymphocytic infiltration of the mucosa with fibrosis and thickening of the muscular layer. The smaller viscus showed less intensive infiltration and less thickening with very little fibrosis.

*Case 3.* This patient, a 51 year old, married woman, complained of a choking sensation and mild pain in the right upper quadrant. These symptoms were of many years duration. She was a tense, nervous individual and her family physician was of the opinion that her complaints were functional.

Cholecystogram showed a functioning gallbladder with an accessory dye filled pouch located medial to the normally situated viscus. These identical findings were present in roentgenologic studies made eight years previous to the one described above. The roentgenologic diagnosis was probably double gallbladder. This patient was not operated upon since it was not believed that her symptoms were due to her gallbladder anomaly.

*Case 4.* This patient was a middle aged woman whose chief complaints were obesity and shortness of breath. There were no symptoms or signs referable to the gallbladder. Gallbladder roentgenologic studies were done as part of a routine examination. The films showed two distinct shadows in the right upper quadrant. These shadows appeared to be connected in the region of the cystic duct. One shadow was somewhat denser than the other and there were no visible stones. The impression was normally functioning double gallbladder. Since none of the patient's difficulty was referable to her biliary system, operation was not advised.

*Bilobed gallbladder:* This anomaly, which Boyden found to be most frequently encountered in cats, is rare in man. As its name implies, it is a gallbladder with a bifid structure. The group of bilobed gallbladders includes those cases in which two gallbladder cavities are drained by a common cystic duct.

There are two different types of this condition. In the first the gallbladder has a longitudinal septum which divides its cavity into two chambers. These chambers communicate at their proximal end and are supplied by one duct. In the second form there is a complete division of the fundic portion into two separate lobes. These two portions fuse at the neck of the gallbladder, forming a common cavity in this region. Thus the two cavities, which are completely separate in their fundic portion, communicate at their base and are drained through a common cystic duct. The lobes are usually about the same size but occasionally one may be considerably larger than the other.

Boyden has demonstrated the embryologic origin of bilobed gallbladders in the cat and it is probable that the same explanation applies to man. In the cat there is frequently a splitting of the gallbladder anlage. This changes a single bud into a bilobed one. These buds maintain a connection at their base but their fundic portions remain separated. In the bilobed organ, in which the two cavities are separated by only a membranous septum, there is probably only an incomplete division of the primitive anlage. When the cavity is then established, a fibrous membrane persists in the area of incomplete separation.

Gross found six anomalies of this type in the literature and we have not been able to find any more reports since his paper was published. We present the roentgenogram of a patient who, we believe, has a bilobed gallbladder. Since there has been no operation, the diagnosis is only presumptive.

*Case 5.* This patient, a 45 year old man, complained of mild nonradiating pain between his scapulae of five years duration. The pain was not constant and did not bear any relation to food. It did not interfere with the performance of his ordinary duties. Physical examination was unimportant except for mild right upper quadrant tenderness.

Cholecystograms showed a good concentration of the opaque media. The gallbladder was doubled on itself and presented the appearance of a bilobed structure (fig. 4). This picture might possibly be produced by a gallbladder with a long overhanging fundus, but we believe that the former diagnosis is by far the more probable.

*Diverticulum of the gallbladder:* Courvoisier[7] found 28 cases of this condition in the literature in 1890 and credited von Heister with having presented the first description in 1717. Very few cases have been seen at operation, probably because they do not produce symptoms.

The diverticula may occur anywhere along the surface of the gallbladder from

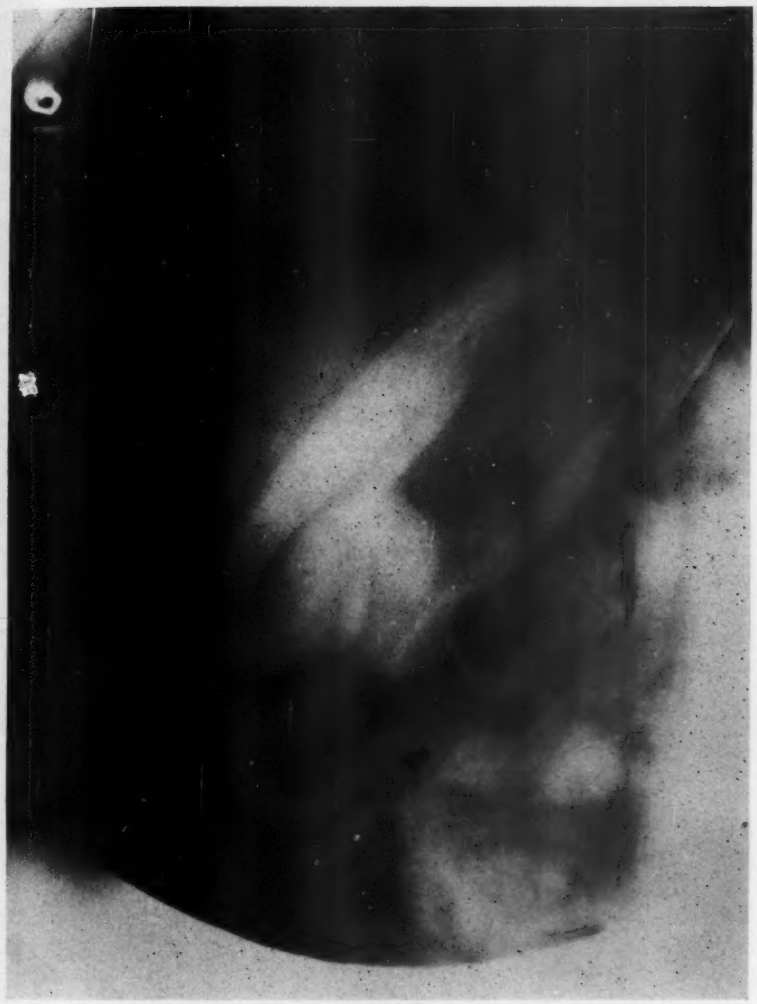

FIG. 4. Cholecystogram of case 5 which appears to show a bilobed gallbladder

the fundus to the neck and they may vary greatly in size. The roentgenologic appearance of this condition has been described by Barsony and Fredrich, Hartman[13] and others and consists of a rounded, saccular area which appears to be tied off from the rest of the gallbladder shadow. The concentrating power of the viscus is usually unimpaired and it empties normally after a fatty meal. Vastine[25] has pointed out that a calculus in a diverticulum can be detected by its separation from, and by its constant relation to the gallbladder shadow. Most diverticula of the gallbladder, particularly those located in the region of

the neck, appear to be related to the development of the cysto-hepatic ducts. These ducts in embryonic life pass from the gallbladder bud or the upper portion of the cystic duct over to the substance of the liver. In the fully formed organ these ducts, when they persist, are found extending from the neck of the gallbladder to the adjacent liver tissue. They rarely are found in human beings.

Although the ducts, and any early diverticula which result from them, would ordinarily be on the side of the gallbladder adjacent to the liver, the normal

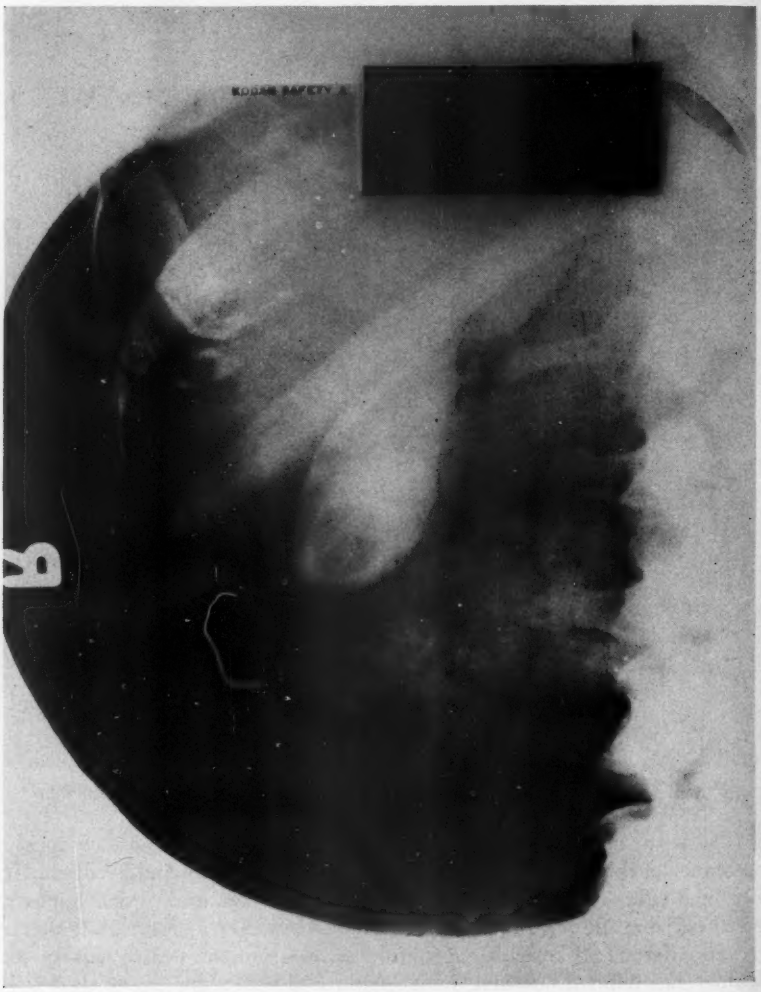

FIG. 5. Cholecystogram of case 6 showing a diverticulum of fundus and large, nonopaque stone in the gallbladder.

rotation of the gallbladder may cause the diverticulum to be on the exposed surface of the organ.

Diverticula of the fundus of the gallbladder cannot be explained in this manner. They are believed to be due to an incomplete resolution of the solid stage through which the gallbladder passes in embryonic life. This results in the formation of a constricting band or an incomplete diaphragm by a process similar to that which produces a congenital stenosis or atresia in the small bowel.

*Case 6.* This patient, a 43 year old white woman, was first seen by her physician for complaints referable to her gallbladder in September 1950. She was suffering from nausea and vomiting which usually followed the ingestion of fatty foods. For 15 years she had had attacks of right upper quadrant pain with radiation around the right costal margin and to the region of the right shoulder.

A cholecystogram revealed a normally functioning gallbladder with a diverticulum of the fundus. The gallbladder contained a large nonopaque stone (fig. 5). This patient did not return to her physician after the cholecystogram was made.

*Case 7.* This patient, a 51 year old married woman, gave a history extending over six years of repeated attacks of epigastric discomfort, bloating, belching and sour eructations. There had been occasional vomiting after meals. Postprandial pain and discomfort usually occurred after the ingestion of fatty foods. Symptoms had been much more severe for the six months previous to admission.

A cholecystogram taken elsewhere one week before admission showed the presence of stones in the gallbladder.

She was a slim, middle-aged woman in no particular distress. There was no icterus. The abdomen was soft and there was tenderness, but no spasm in the right upper quadrant.

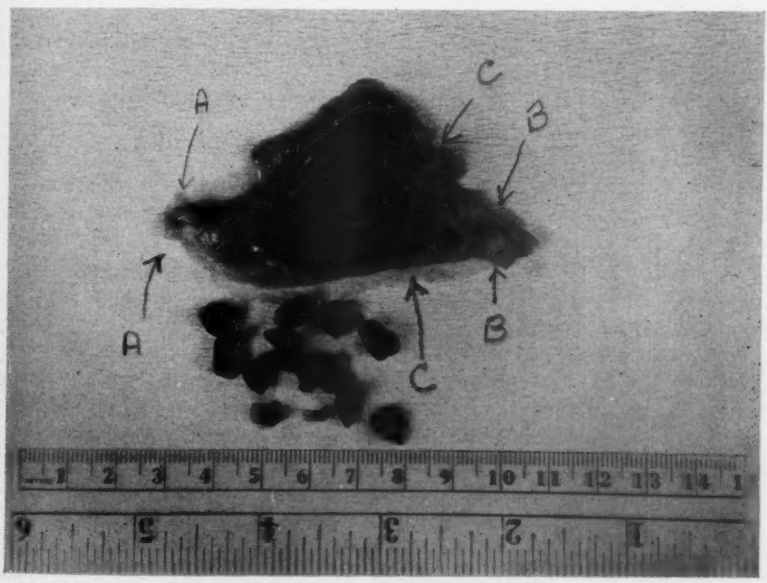


FIG. 6. Surgical specimen, case 7. A. cystic duct; B. diverticulum; C. membrane

There was also tenderness in the right costovertebral area. The right kidney was palpable and quite mobile.

At operation, a normal sized, thickened gallbladder was found, which contained many small stones. At its fundus there was a small diverticulum which contained two small stones. When the viscus was opened, an incomplete membrane or diaphragm was found to extend across the upper part of the gallbladder, proximal to the mouth of the diverticulum (fig. 6). Microscopic examination showed edema with fibrosis and some hyalinization. There were areas of lymphocytic infiltration. The pathologic diagnosis was chronically inflamed diverticulum.

## ANOMALIES OF POSITION

Abnormally located gallbladders are rarely encountered, but their more usual ectopic locations should be known and the technical difficulties associated with them understood.

*Left sided gallbladder:* In all the recorded cases of this type reviewed by Gross, the viscus laid to the left of the falciform ligament; if this structure is broad, it may be hidden from view when the ordinary right upper quadrant approach is employed. The cystic duct may enter the right hepatic duct or at the junction of the hepatic and common ducts.

Two possible embryologic origins for left sided gallbladders have been advanced and seem to be necessary to explain the two different ductal arrangements mentioned above. The left sided gallbladder which has its cystic duct emptying into the junction of the common and hepatic ducts probably arises as a normal gallbladder bud springing from the hepatic diverticulum. Instead of migrating to the right, however, this bud migrates to the left and takes up its position beneath the left lobe of the liver. In the cases described by Harris[12] and by Walton[27] the cystic duct arose from the junction of the common and hepatic ducts and was at first directed toward the right. It then turned sharply and passed to the left dipping behind the falciform ligament and running to the gallbladder beneath the left lobe of the liver. Such an arrangement fits in admirably with the above description.

A different mode of abnormal development must be sought to explain the origin of those left-sided gallbladders in which the cystic duct drains into the left hepatic duct. This type of case was described by Kehr. It is believed that here we have an independent development of a second gallbladder bud directly from the left hepatic duct and that this is accompanied by an agenesis of the normal structure on the right side.

*Intrahepatic gallbladder:* The true intrahepatic gallbladder lies wholly within the substance of the liver so that at first glance it may seem to be absent. Its position may be betrayed by a bulge of the hepatic tissue on the under surface of the liver or a small portion of the neck may be visible. In some cases, as in the one reported by Stetten,[24] the fundus may protrude from the superior surface of the liver. This author believes that an intrahepatic gallbladder containing stones is best treated by opening the viscus, removing the stones and instituting drainage. In this manner the hemorrhage which would inevitably accompany removal of this type of gallbladder can be avoided. Variations of the true intra-

hepatic gallbladder are seen in the viscus which is entirely exposed except for a bridge of hepatic tissue which completely encircles a portion of its body. The fundus of the gallbladder may in some cases be completely buried in hepatic tissue while the remainder of the organ is exposed. All surgeons are familiar with the rather frequently encountered imbedded gallbladder in which only a portion of the under surface is exposed.

Embryologically the intrahepatic gallbladder is believed to be a congenital arrest since, during the second month of fetal life, this viscus is buried in hepatic tissue and becomes superficial at a later date.

*Retrodisplacement of the gallbladder:* In this anomaly the cystic duct arises from the usual position but the gallbladder, instead of being situated on the under surface of the right lobe of the liver, is directed upward and backward and lies on the inferior and posterior surface of the right lobe. The difficulties facing the surgeon who attempts to remove this type of gallbladder are obvious.

Retrodisplaced gallbladders are believed to be due to a migration of the gallbladder bud from its normal course to final fixation in the position described above.

*Transverse position of the gallbladder:* Gross found 2 cases of this type in his review of the literature. In both of these cases the gallbladder lay in a horizontal position resting in the transverse fissure. In 1 of the 2 cases the gallbladder was about one-third the normal size.

This anomaly is also believed to arise congenitally from a malrotation of the gallbladder bud.

*Floating gallbladder:* This is the most frequently encountered anomaly of the gallbladder and the one which has the greatest surgical significance. In such cases the gallbladder has a mesentery of varying length which permits the organ to hang down from the inferior hepatic surface. This mesentery may run the entire length of the organ supporting it and the cystic duct, or only the cystic duct may be supported, the gallbladder itself hanging free in the peritoneal cavity. The incidence of this condition according to Gross has been listed as high as 4 to 5 per cent of dissecting room specimens.

The surgical significance of this anomaly lies in the fact that these mobile gallbladders may become twisted, producing tortion and obstruction of their blood supply with subsequent infarction. This surgical emergency is most frequently seen in women over 50 years of age, the patients usually being the ptotic type individuals. There is an abrupt onset of intense, constant pain in the right upper quadrant. This pain does not radiate. There is nausea and vomiting and collapse may occur. The temperature is rarely over 100 F., but the pulse rate is usually rapid and may range as high as 150. Leukocytosis is the rule. In the great majority of cases there is no history of a previous attack.

Examination will usually reveal tenderness and spasm in the right upper quadrant. The affected gallbladder may be palpable and may be felt in its normal position or, due to its mobility, in other parts of the abdomen.

At operation tortion of the mesentery and cystic artery with varying stages of infarction of the gallbladder are found. The nature of the anomaly makes surgi-

cal treatment remarkably simple. The cystic duct is easily clamped, divided and ligated and the presence of the mesentery enables one to remove the gallbladder with a minimum of dissection. Recovery is the rule if operation is done before perforation occurs.

### ABSENCE OF THE GALLBLADDER

Mellville[19] in 1937 found 38 cases of this anomaly recorded in the literature and in 1945 Dixon and Lichtman[9] added 10 more. These were cases in which absence of the gallbladder was the only abnormality of the biliary system. The condition is much more frequent if we consider those cases which are associated with atresia of one of the hepatic ducts or the common duct. In about one-sixth of the cases of atresia of the extra-hepatic biliary system there is absence of the gallbladder.

In many of the reports the quadrate lobe has been either absent or poorly defined. As far as can be determined the absence of the gallbladder per se does not impair the health of the individual and the presence of this anomaly is not accompanied by any compensatory dilatation of the bile ducts.

Absence of the gallbladder may be explained embryologically in two ways. Normally this organ and the cystic duct originate as an out-pouching of the hepatic diverticulum, which in turn originates as an outgrowth from the foregut. The failure of this out-pouching to develop from the hepatic diverticulum would naturally result in a complete absence of the gallbladder and cystic duct. A second explanation for the occurrence of some of these cases is an arrest in the development of the gallbladder before it forms its final hollow structure. The extra-hepatic ductal structures, as well as the gallbladder, are hollow in early embryonic life, but later their lumens are obliterated during the solid phase of their development. Still later the lumens are re-established. An arrest in development of any portion of the ductal system during the solid stage will produce an atresia at this point in the same way that atresias of the intestinal tract are produced. When this type of arrest develops in the gallbladder anlage there remains a solid cord representing the gallbladder and cystic duct. This is the type of anomaly usually found associated with atresia of the extra-hepatic ductal system.

*Case 8:* This patient, a 49 year old Negro woman, was admitted to the Norfolk General Hospital with a history of indigestion and intolerance to fatty foods of one year duration. Six weeks before admission she had an attack of sharp right upper quadrant pain and fullness which came on after a meal of fried oysters. Following this attack, the patient noticed that her sclera and skin turned yellow. This discoloration persisted. She also noticed that her urine became yellow and that her stools were light. She had two more similar attacks before admission to the hospital. System review and past history were noncontributory.

Physical examination showed slight icterus of the sclera and skin. The blood pressure was 200/100. There was slight tenderness in the right upper quadrant. No masses were palpable. The icteric index was 55. Urinary urobilinogen was present and there was urobilinogen in the stool.

A diagnosis of common duct stone was made and exploration was done. The transverse colon was found to be adherent to the under surface of the liver in the area normally occupied by the gallbladder. The duodenum and pyloric portion of the stomach were also in-

volved in these adhesions. When these structures were freed, no gallbladder was found. The common duct was dilated and there was a small nodule or outpocketing at the site where the origin of the cystic duct would ordinarily be located.

The common duct was opened and found to contain purulent bile. A stone about 0.5 by 2.0 cm. in size was located in the ampulla of Vater, it was milked back into the duct and removed. T tube drainage of the common duct was instituted. Tissue for biopsy was taken from the liver. The liver biopsy showed bile stasis and chronic hepatitis.

Eleven days after the operation, lipiodol was injected into the T tube and a cholangiogram showed that the dye readily ran into the duodenum. The hepatic ducts appeared normal, the left being slightly larger than the right. There was no evidence of a cystic duct or gallbladder. On the thirteenth postoperative day, a cholecystogram did not reveal any gallbladder visualization. This patient's postoperative course was uneventful. The symptoms disappeared and the significant preoperative laboratory findings gradually returned to normal.

### OTHER ABNORMALITIES OF THE GALLBLADDER

A Phrygian cap is an apparent angulation of the distal portion of the fundus of the gallbladder. This appearance is produced by a septum located at the point

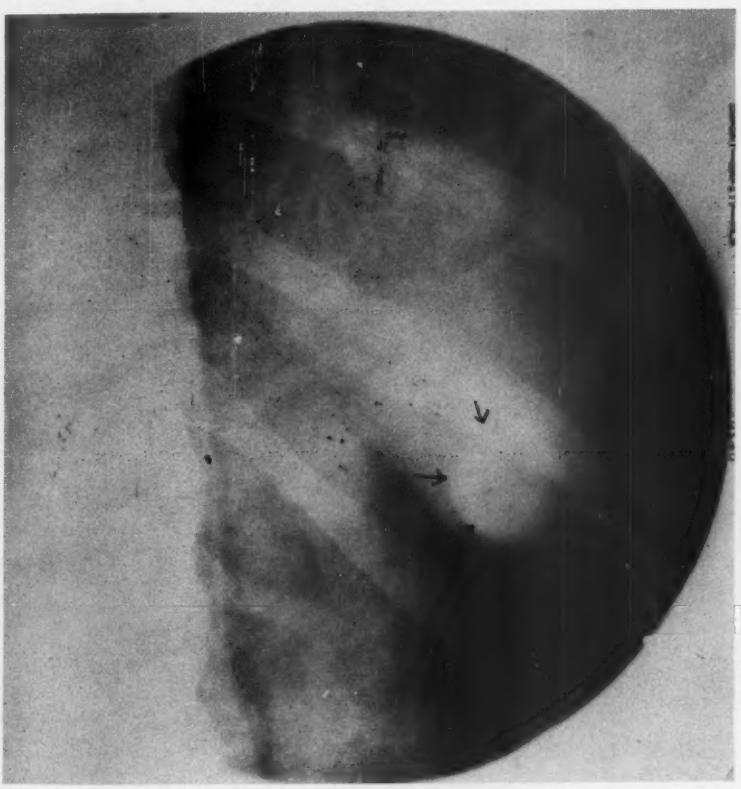

FIG. 7. Phrygian cap simulating stone in the gallbladder

of apparent folding which extends into the lumen of the gallbladder and produces various degrees of biloculation. The condition was first reported by Bartel[3] in 1916. He chose the name "Phrygian Cap" because of its resemblance to the hats worn by the people of Phrygia, an ancient country of Asia. The goddess of liberty is still occasionally pictured wearing this type of headgear. This condition is not rare and is seen frequently in cholecystograms. It is often overlooked at operation. It was previously thought to be of definite clinical significance, but Boyden[4] in 1935 made a careful study of it and reported a series of

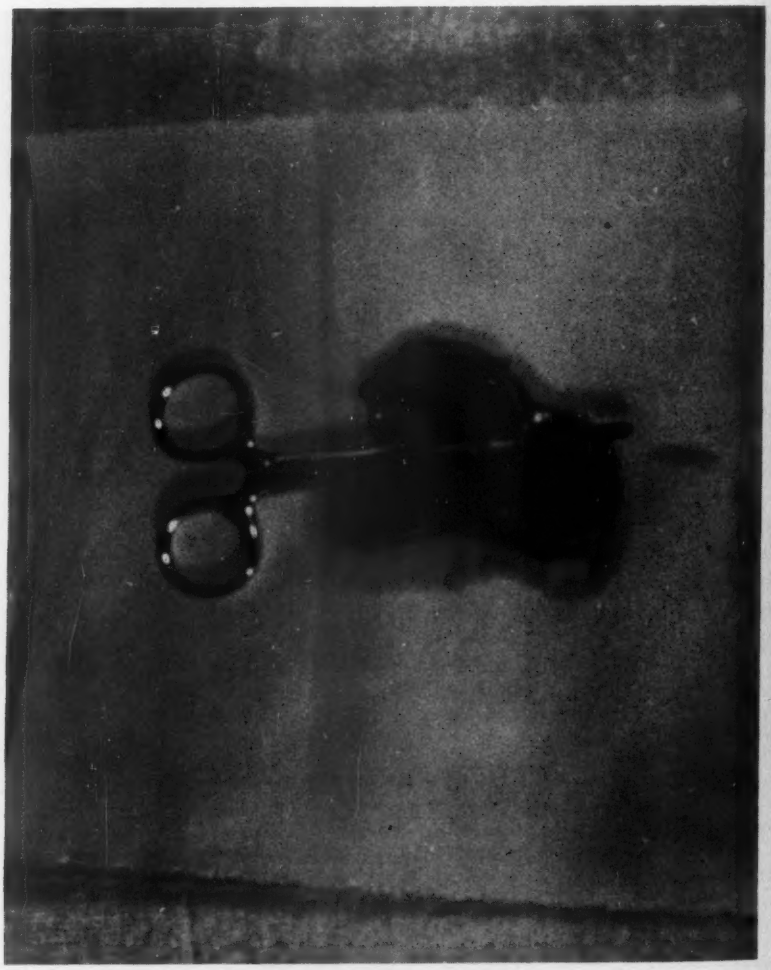

FIG. 8. Septum in lower portion of gallbladder dividing the viscus into two distinct compartments. Tip of hemostat is inserted through communicating sinus.

165 cholecystograms of normal persons in which the incidence of Phrygian cap was 3.6 per cent. Meyer, Carter and Mecker[21] reported an incidence of 4 per cent in routine cholecystograms and 6 per cent in a series of gallbladders removed at operation. The concensus of opinion today seems to be that this condition in itself is of no clinical importance except that it may simulate a stone on roentgenologic examination (fig. 7).

The fact that the Phrygian cap is a congenital and not an acquired variation has been well demonstrated by Boyden and Cesarina.[5] The presence of a septum in the gallbladder is believed to be due to incomplete vacuolization of the solid

FIG. 9. Hourglass deformity of the gallbladder

gallbladder bud when this structure goes through the process which re-establishes its lumen.

Septums which are believed to be congenital and which probably develop by the same process may be found in other parts of the gallbladder. The septum or membrane which is often associated with a diverticulum has been mentioned above and illustrated in figure 6. Figure 8 shows a septum which is located in the lower part of the gallbladder and divides the viscus into two definite compartments. This is a surgical specimen obtained from a middle-aged Negro woman who had suffered from typical gallbladder colic and fatty food intolerance for several months. Before the septum was divided, the only communication between the two compartments was a small aperture less than ½ cm. in diameter. Both compartments were filled with small stones and gravel. A cholecystogram taken before operation did not visualize the gallbladder.

Hour glass deformity of the gallbladder is described frequently, but it cannot be determined from most reports whether the condition is congenital or acquired (fig. 9).

Gross believes that congenital forms of this condition do exist and are apt to be associated with atresia or other malformations of the bile ducts.

Rudimentary gallbladders exist also, but like the above condition, the true congenital form is often confused with small contracted gallbladders resulting from scar formation. Congenital rudimentary gallbladders are usually found in infants and children and represent a congenital hypoplasia.

Persistent cysto-hepatic ducts, although not true congenital anomalies of the gallbladder, should be mentioned here because they are surgically significant and are not ordinarily mentioned in discussions of anomalies of the extra-hepatic ductal system. As mentioned in the discussion of the formation of diverticuli of the gallbladder, the cysto-hepatic ducts are structures which are normally present in embryonic life and extend from the upper end of the cystic duct and the lower portion of the gallbladder directly into the liver substance. Their persistence in adult life is rare but they should be looked for in every cholecystectomy and ligated when found. Failure to do this will result in profuse, persistent postoperative biliary drainage and will increase the incidence of biliary peritonitis.

SUMMARY

Congenital anomalies of the gallbladder are discussed. A classification of these conditions is presented and their embryologic origins are considered. The characteristic roentgenographic appearance of each anomaly is described and its clinic and surgical significance emphasized.

REFERENCES

1. Barsony, T., and von Friedrich, L.: Divertikel der gallenblase, Klin. Wchnschr. *7:* 216 (Jan. 29) 1928.
2. Bartel, J.: Cholelithiasis und korperhonstitution, Frankfurt Ztschr. Path. *19:* 206, 1916.
3. Boyden, E. A.: Accessory gallbladder: embryological and comparative study of aberrant biliary vesicles occurring in man and domestic animals, Am. J. Anat. *38:* 177 (Nov.) 1926.

4. Boyden, E. A.: Phrygian cap in cholecystography, Am. J. Roentgenol. *33:* 589 (May) 1935.
5. Cesarini, M.: Contributo allo studio delle deformazioni delle colecisti, Quaderni radiol. *6:* 9, 1935.
6. Climan, M.: Duplication of gallbladder demonstrated by cholecystography, M. J. & Rec. *130:* 73 (July 17) 1929.
7. Courvoisier, Z. G.: Casuistisch-statistische beitrage zur pathologie und chirurgie der gallenwege, p. 12 Leipzig F. C. W. Vogel, 1890.
8. Croudace, W. H. H.: Case of double gallbladder, Brit. M. J. *1:* 707 (April 25) 1931.
9. Dixon, C. F., and Lightman, A. L.: Congenital absence of gall bladder, Surgery *17:* 11 (Jan.) 1945.
10. Eisendrath, D. N.: Anomalies of bile ducts and blood vessels, J.A.M.A. *71:* 864 (Sept. 14) 1918.
11. Gross, R. E.: Congenital anomalies of gallbladder; review of 148 cases, with report of double gallbladder, Arch. Surg. *32:* 131 (Jan.) 1936.
12. Harris, H. A.: Intrahepatic ectopic testis combined with ectopic vesical, congenital umbilical hernia and abnormal gallbladder, Arch. Surg. *13:* 644 (Nov.) 1926.
13. Hartman, H.: Quelques points de l'anatomie et de la chirurgie des voies biliaires, Bull. Soc. anat. de Paris *5:* 480 (July) 1891.
14. Holmes, J. B.: Congenital obliteration of bile ducts, Am. J. Dis. Child. *2:* 405 (June) 1916.
15. Howard, C. P., and Wolback, S. B.: Congenital obliteration of bile ducts, Arch. Int. Med. *8:* 557 (Dec.) 1911.
16. Kehr, H.: Eine seltene anomalie der gallengange, Munchen. med. Wchnschr. *49:* 229 (Feb. 11) 1902.
17. Kiebel and Mall: Human Embryology, Philadelphia, J. B. Lippincott Co. *2:* 413, 1912.
18. Ladd, W. E.: Congenital atresia and stenosis of bile ducts, J.A.M.A. *91:* 1082 (Oct. 13) 1928.
19. Melville, A. G. G.: Case of absence of gall bladder and duodenal diverticulosis, Acta radiol. *18:* 65, 1937.
20. Mentzer, S. H.: Anomalous bile ducts in man, J.A.M.A. *93:* 1273 (Oct. 26) 1929.
21. Meyer, W. H., Carter, R. F., and Meeker, L. H.: "Phrygian cap" deformity of gallbladder, Am. J. Roentgenol. *37:* 786 (June) 1937.
22. Nichols, B. H.: Double gallbladder, Radiol. *6:* 255 (March) 1926.
23. Sherren, J.: Double gallbladder removed by operation, Ann. Surg. *54:* 204 (Aug.) 1911.
24. Stetten, DeW.: Cholecystectomy in case of intrahepatic gallbladder, Ann. Surg. *97:* 296 (Feb.) 1933.
25. Vastine, J. H.: Diverticulum of gallbladder, Am. J. Roentgenol. *31:* 603 (May) 1934.
26. Verstandig, C. C., and Moore, D. B.: Double gall bladder demonstrated by oral cholecystography, Connecticut M. J. *12:* 314 (April) 1948.
27. Walton, A. J.: Congenital malposition of gallbladder, Lancet *1:* 925 (April 6) 1912.

# TRANS-STERNAL ANTERIOR MEDIASTINAL DISSECTION FOR CANCER OF THE THYROID

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## INTRODUCTION

Cancer of the thyroid gland varies considerably in its architectural pattern, its degree of anaplasia, rapidity of growth and mode of dissemination. These factors make it extremely difficult to reach a decision regarding the range of surgical resection necessary in any patient. Papillary carcinoma of the thyroid, because of its predilection to enter lymphatics, can, when metastases are present, be most adequately removed by a combination of trans-sternal anterior mediastinal and radical neck dissection. Fortunately, this group comprises the largest numerical division of thyroid malignancies. Adenocarcinomas of the thyroid on the other hand usually metastasize via the blood stream, and are less apt to receive benefit from radical extirpative surgery. There are exceptions, however, in that a bulky malignant lesion arising in the thyroid that is causing tracheal compression or the inferior borders of which extend too deeply into the anterior mediastinum to be easily approached from the cervical incision alone, will be more safely removed through a sternal approach. Under these circumstances, where there is cardio-respiratory embarrassment, the improvement obtained by relieving the obstruction affords the patient a very satisfactory measure of palliation. Furthermore, in large adenocarcinomas, the removal of the main mass of malignant tissue and all of the thyroid gland will greatly enhance postoperative radiation therapy whether it be from the roentgen ray or by radioactive isotopes.

At the present time combined mediastinal and radical neck dissection is not considered necessary for those patients from whom a single nodule of the thyroid has been excised and the pathologist is the first to establish a diagnosis of malignancy. This arbitrary point of view has been tentatively adopted in an attempt to determine the necessity of doing radical surgery for this problem. If, however, such a tumor recurs locally or metastases appear in the cervical lymphatics, then the complete area of lymphatic drainage from the thyroid requires resection. The extension of this lymphatic network into the anterior mediastinum has long been known, but all too frequently disregarded in the design of operative procedures for the excision of thyroid cancer.

King[6] and Sir Astley Cooper[2] in 1836 directed attention to the retrosternal portion of the lymphatics draining the thyroid. Much of our present knowledge concerning the lymph drainage from the thyroid gland stems from investigations

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Presented during the Birmingham Assembly of The Southeastern Surgical Congress, March 8–11, 1954, Birmingham, Ala.

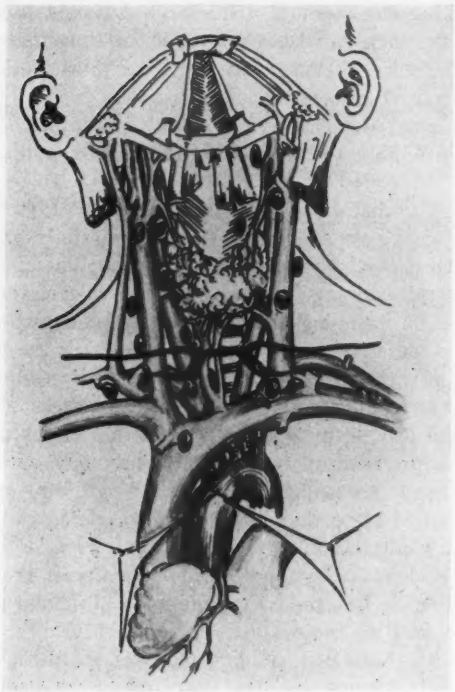


FIG. 1. Distribution of lymph nodes draining the thyroid gland. Metastases have been found in all locations.

carried out between 1927 and 1938. Rouvière[10] in his study of lymphatics published in 1938, described inferior collection channels which drain into nodes at the angle of the two innominate veins. Other studies have shown the existence of direct, uninterrupted channels from the thyroid to the junction of the internal jugular with the subclavian vein.[7] Some lymph vessels extend downward from the thyroid gland to the level of the innominate veins and they may be interrupted at any point by nodes.[1, 4] Such lymphatics may occur anywhere between the two internal jugular veins. These inferior groups have not received the emphasis which the middle and superior cervical groups of lymph nodes have had in planning the excision of thyroid cancer. All of these groups are depicted by single lymph nodes in figure 1.

## TECHNIC

The principle of en bloc excision of a cancer-bearing organ and its lymphatic drainage is well established. McClintock and his colleagues[8] and McCorkle et al[9] have used this largely forgotten knowledge of thyroid lymphatic anatomy in their application of en bloc excision to malignant disease of the thyroid. To fulfill the requirements of this principle, access to the anterior mediastinum is

obtained by splitting the sternum. The cervical area is usually approached through a high collar incision with exposure of the sternum by a vertical component down to the third interspace. Usually, the third costal cartilage on the side of principal thyroid involvement is removed to permit blunt finger dissection of the retrosternal tissue away from the manubrium. This bone is then split with the Lebsche knife to the level of the removed cartilage where one-half of the sternum is transected.

The anterior mediastinal dissection has included the thymus and fat pad in this area.* The superior vena cava is stripped anteriorly and laterally to its bifurcation and the dissection is carried around the innominate veins to their junction with the internal jugular veins. On the side of the primary tumor, radical neck dissection including muscles, veins and homolateral thyroid lobe is made en bloc. The common carotid artery is preserved; the phrenic and vagus nerves are spared as is the recurrent laryngeal nerve, if possible. This procedure has the advantage of making it easy to identify the nerves and vessels, but has the disadvantage of leaving until last the dissection of the primarily involved thyroid lobe. Thus, invasion of larynx or trachea may not be revealed until late in the operation. The addition of total laryngectomy or partial tracheal resection are operative steps that have been considered but not employed in the cases reported. Contralateral thyroid lobectomy is done. If grossly involved lymph nodes are evident in the opposite side of the neck, they can be removed by a modified neck dissection preserving the internal jugular vein if done at the primary operation, or their removal can be deferred for a secondary procedure.

This operation has been done under endotracheal nitrous-oxide anesthesia supplemented by intravenous pentathol and barbiturate medication. Blood loss is measured gravimetrically and replaced as it is lost during the operative procedure. If the pleural cavity is inadvertently opened, catheter drainage is instituted at the close of the operation. The mediastinum is drained through a separate catheter. Usually, the lateral angle of the cervical incision is also drained. If prophylactic tracheostomy is done at the conclusion of the procedure, the subcutaneous tissues are sutured to the trachea before it is opened to make an air tight seal around the tube. This has prevented mediastinal emphysema and infection.

## CASE REPORTS

*Case 1.* (Case No. 10 of series) B. L., a 24 year old woman was admitted to the Albany Hospital on Dec. 11, 1953. In the course of a routine employment examination six weeks prior to her admission, a nodule was discovered in her thyroid gland.

On physical examination a rounded swelling was visible in the episternal notch emerging from behind the left sternoclavicular joint. On palpation this mass seemed to be cystic and was completely separate from the palpably normal thyroid.

Operation on Dec. 12, 1953, began as an excision of what was thought might be a cystic hygroma. After it was exposed, the gross appearance of this mass was typical of papillary thyroid carcinoma metastatic in a lymph node. Examination of the inferior pole of the left

* No cancer bearing lymph nodes have been found inferior to the innominate veins in our series.

thyroid lobe led to the discovery of a similar mass 1 cm. in diameter which was hidden under the pole but completely separated from it. This small nodule was sent to the laboratory from where a frozen section diagnosis of "adenomatous thyroid tissue" was returned. A tiny cystic nodule was present in the left part of the thyroid isthmus (fig. 2).

On the basis of the gross appearance and histologic findings, the patient was redraped and a trans-sternal mediastinal and left radical neck dissection with total thyroidectomy was done. The large, extra-thyroidal mass rested in the angle of the left internal jugular with its innominate vein.

The pathologist stated that, although in some places the adenomata grew as papillary cystadenomata, from a histologic standpoint, no definite evidence of malignancy could be found in the sections studied. The fact that adenomatous and papillary cystadenomatous thyroid tissue was found totally apart from the thyroid gland, both in the anterior medias-

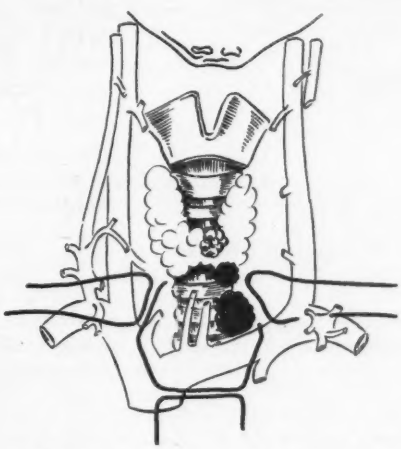


FIG. 2. Case 1. Cystic nodule isthmus and separated masses of adenomatous thyroid with papillary changes.

tinum and under the left inferior pole of the thyroid would suggest that, in spite of the histologic innocence of the lesion, careful consideration should be given to a diagnosis of papillary cystadenocarcinoma of thyroid origin.

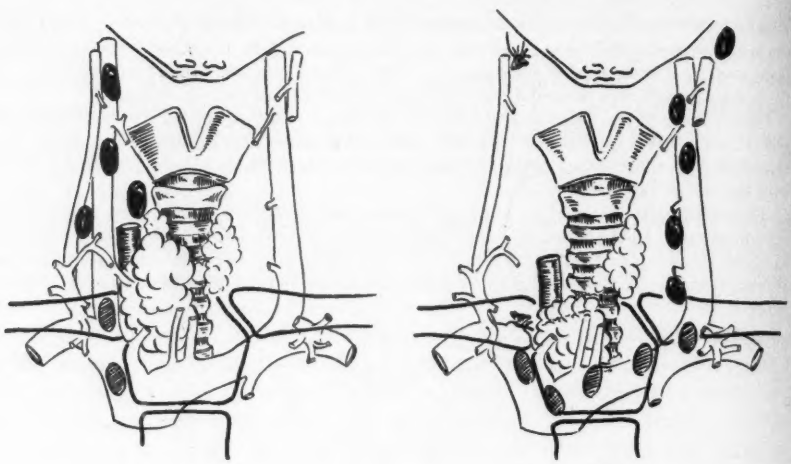
The patient returned to her duties as a school teacher in January, fortified by 1 grain of desiccated thyroid each day.

*Case 2.* (Case No. 11 of series) D. Y., a 17 year old girl was admitted to the Albany Hospital on July 27, 1953, because of an enlarging mass in the base of the neck on the left side.

The past history included right thyroid lobectomy and radical neck dissection for adenocarcinoma of the thyroid on Nov. 29, 1947, by the senior author, (fig. 3a). Further operative removal of her tumor was attempted on the left side in Memorial Hospital, New York, followed by treatment with radioactive iodine. Because of this experience the patient is included in the report of Duffy and Fitzgerald (Case No. 28).[3]

Physical examination disclosed a hard, irregular fixed mass just above and to the left of the sternum. There were many hard lymph nodes present under the left sternocleidomastoid muscle, (fig. 3b). Roentgenographic studies of the chest revealed a nodular shadow 7 mm. in diameter in the third right anterior interspace.

Operation consisting of trans-sternal anterior mediastinal and left radical neck dissection with left thyroid lobectomy and tracheostomy was done on July 29, 1953. The tracheos-



3A 3B

FIG. 3A. Case 2. Original right sided tumor and lymph node metastases
FIG. 3B. Case 2. Lymph node metastases found at operation in 1953

tomy tube was removed on August 15. Infection of the sternal wound was noted on August 6, and this necessitated removal on Jan. 29, 1954, of the subcutaneous silk sutures used to close the wound.

Pathologic report showed fibro-adipose tissue replacing the left thyroid lobe. Metastatic adenocarcinoma was demonstrated in the superior, middle and inferior cervical lymph nodes and in nodes adjacent to the innominate veins. The metastases were histologically identical with the original tumor which was removed from the right thyroid lobe in 1947.

In February 1954 this patient was living without evidence of any progression in the pulmonary lesion. She is being prepared for $I^{131}$ studies to determine future therapy.

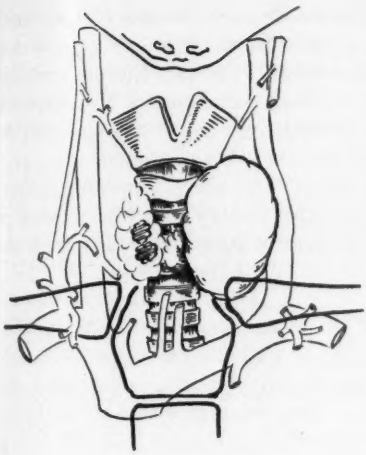
*Case 3.* (Case No. 12 of series) A. M. K., a 34 year old woman was admitted to the Albany Hospital on May 17, 1953. A small, hard nodule had been noted 10 days before in the right side of the neck at the inferior border of the larynx.

The past history included bilateral subtotal thyroidectomy at the age of 16 in 1935. The pathologic report stated in part. . . . "The hyperplastic nodules are so atypical that they seem almost malignant but no mitotic figures or intravascular masses are found. . . ." (fig. 4a). The second admission in 1939 was because of a left sided recurrence of one year's duration. Left thyroid lobectomy was done on Jan. 13, 1939. The pathologic report was "Malignant adenoma of thyroid gland", (fig. 4b). Postoperatively, 2550 r measured in air were delivered through an anterior neck port. The third admission was in 1946 for a recurrent nodule in the isthmus and right thyroid lobe. This was removed on June 3, 1946, with the overlying sternothyroid muscle. Pathology was again reported as malignant adenoma of the thyroid gland (fig. 4c). Postoperative radiation treatment, totalling 5600 measured in air, was administered through the four ports: right and left lateral neck; anterior and posterior, superior mediastinum.

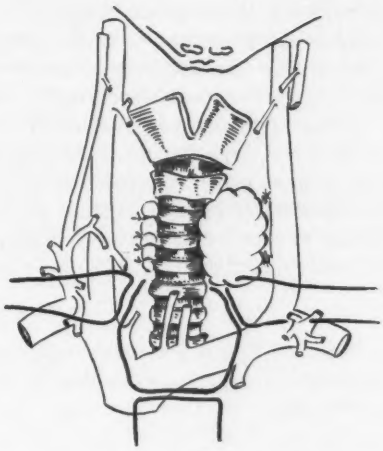
The physical examination in May 1953, revealed a small, hard fixed nodule on the right side of the trachea at the inferior border of the larynx.

Operation consisting of trans-sternal anterior mediastinal and right radical neck dissection was done on May 19, 1953. Tracheostomy was not required.

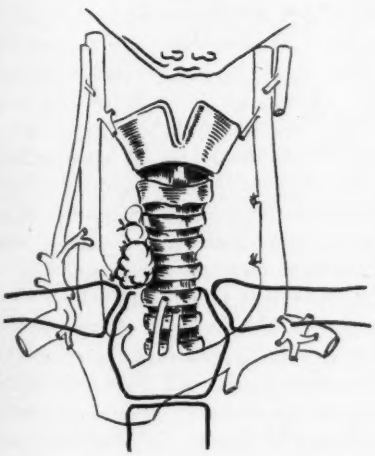
The pathologic report showed adenocarcinoma of the thyroid gland was found only in the recurrence on the trachea (fig. 4d). All lymphatic structures were found to be free of tumor.



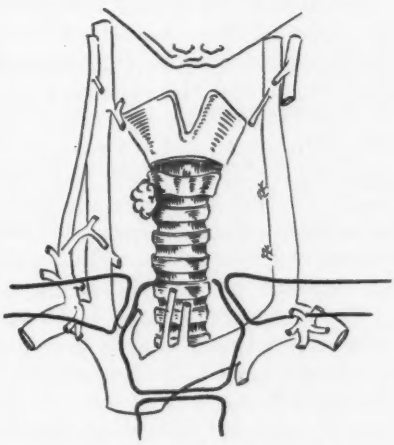
4A



4B



4C



4D

FIG. 4A. Case 3. Original cyst left thyroid lobe and nodules in right lobe
FIG. 4B. Case 3. Left sided recurrence 1939
FIG. 4C. Case 3. Right sided recurrence 1946
FIG. 4D. Case 3. Right sided recurrence 1953. No lymph node metastases at any time

The radiation damaged area of the skin on the right allowed wound separation of the collar portion for a distance of 3 inches. This healed by secondary intention.

In February 1954, this patient was living with no demonstrable evidence of her malignant disease.

## DISCUSSION

Of the 3 cases reported, the first patient has features that are of great interest. Allen Graham[5] who graciously reviewed the slides from this case believes that it represents accessory thyroid tissue associated with an old nodular goiter with

no evidence of malignant disease. The absence of any evidence of lymphoid tissue in many sections from the extra-glandular thyroid masses supports this point of view. Because there is controversy about this aberrant thyroid problem the surgeon has great responsibility when deciding the extent of his resection.

The second patient is an example of the benefits it is hoped will accrue from removal of a large amount of thyroid tissue; namely, remaining metastases may now be more easily stimulated to pick up radioactive iodine. The third patient is of particular interest because of the very long history and the absence of distant or even local metastases. This patient has not benefited greatly from the extensive resection.

## RESULTS

Together with 9 previously reported cases, 12 patients have now been subjected to trans-sternal dissection of the anterior mediastinum, radical neck dissection and total thyroidectomy. There has been no postoperative mortality. All patients are living without evidence of malignant disease with the exceptions noted above and as follows:

One patient, case No. 1 of the series[8] is dead from her disease 22 months after the procedure. A second patient, case No. 3 of the series[8] is alive 15 months after operation with recurrence in lymph nodes at the tip of the mastoid, in the mediastinum and with bilateral pulmonary metastases. A third patient, case No. 9 of the series[8] has such extensive laryngeal recurrence that further surgery is inadvisable. All but 1 (case 1 of this report) of these first 12 patients had either very extensive lymph node metastases or were recurrent after previous surgery, so that it was not expected to obtain excellent long term results. It is anticipated that as patients who suffer from early malignant disease of the thyroid with few cervical and no mediastinal node metastases are subjected to the proposed operation, 10 and 20 year follow-up results will improve.

The persistence of tumor in the larynx in 1 patient reported above and the recurrence in lymph nodes in the anterior mediastinum with lung metastases in a second case raise the question of the adequacy of even this wide type of resection. It is believed that more extensive surgery is necessary, if one is to eradicate far advanced cervical extension from cancer of the thyroid. For those patients in whom obvious tumor is observed invading the larynx, immediate laryngectomy may be worth while. If the area of invasion is localized to the trachea, partial tracheal resection may suffice. The second patient with persistence in the mediastinum and the development of new lymph nodes at the tip of the mastoid indicates the necessity for carrying the initial dissection to the base of the skull. It should be borne in mind that these 2 patients came to surgery with very extensive metastatic malignant disease and for them the initial surgery had not included a sufficiently wide margin. Modifications in technic are necessary to give such individuals the maximum hope of relief from their disease.

This procedure appears particularly suitable for that patient with only a few metastases to the middle or inferior cervical lymph node groups. Thus, when

superior cervical and mediastinal nodes are proved histologically to be free of malignant disease, maximum benefit should result. The procedure is designed to make possible wide excision of a cancer bearing organ and all of its lymphatic drainage. The results of this early experience, even in the advanced cases reported above, encourage us to pursue the problem further.

## SUMMARY

A combination of total thyroidectomy, radical neck and anterior mediastinal dissection for the en bloc removal of certain thyroid malignancies is proposed.

The papillary adenocarcinomas will possibly lend themselves best to this mode of surgical therapy.

Three additional cases are added to the 9 previously recorded.

## REFERENCES

1. Chouke, K. S., Whitehead, R. W., and Parker, A. E.: Is there a closed lymphatic system connecting the thyroid and thymus glands, Surg., Gynec. & Obst. *54:* 865 (June) 1952.
2. Cooper, Sir Astley: Notes on thyroid gland, Guys Hosp. Rep. *1:* 456, 1836.
3. Duffy, B. J., Jr., and Fitzgerald, P. J.: Cancer of thyroid in children; report of 28 cases, J. Clin. Endocrinol. *10:* 1296 (Oct.) 1950.
4. Gordon, S. D.: Lymphatic system of thyroid gland, Canad. M.A.J. *25:* 46, 1931.
5. Graham, A.: Personal communication.
6. King, T. W.: Observations on thyroid gland, Guys Hosp. Rep. *1:* 429, 1836.
7. Mahorner, H. R., Caylor, H. D., Schlotthauer, C. F., and Pemberton, J. deJ.: Observations on lymphatic connections of thyroid gland in man, Anat. Rec. *36:* 341 (Oct. 25) 1927.
8. McClintock, J. C., Stranahan, A., Alley, R., and Baker, W.: Thoraco-cervical approach for malignant disease of thyroid, Ann. Surg. *139:* 158 (Feb.) 1954.
9. McCorkle, H. J., Davis, C., Galante, M., and Saunders, J. B.: Dissection of the superior mediastinum for thyroid cancer, A.M.A. Arch. Surg. *66:* 798 (June) 1953.
10. Rouvière, H.: Anatomy of human lymphatic system, Transl. by M. J. Tobias, Ann Arbor, Edward Brothers, Inc., *8:* 63, 1938.

# COLOSTOMY AS EMERGENCY TREATMENT FOR MASSIVE MELENA SECONDARY TO DIVERTICULITIS

## CASE REPORT

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In the past most of the literature about operative procedures for diverticulitis concerned itself with abscess formation, fistulas, obstruction, and inability to distinguish the lesion from carcinoma. Of late there has been some evidence of severe melena resulting from diverticulitis and requiring emergency and massive surgery. One such case was reported by Cate[1] and he was able to find only 1 other case report[3] in which laparotomy was done for the purpose of controlling blood loss. In Cate's case he was confronted with a problem of a massive melena which was not controlled by conservative measures. An emergency laparotomy was done and it failed to reveal any cause of bleeding other than multiple diverticula. The bleeding continued and 18 hours later a colectomy was done followed by iliosigmoidostomy. The patient recovered.

Recently Quinn and Ochsner[2] reported a series of cases of 7 patients operated upon because of massive rectal bleeding. In 1 an emergency laparotomy was done and no bleeding area could be found. Two days later this patient was again explored and no bleeding point could be demonstrated but a total colectomy was done. The patient died on the third postoperative day. In the 6 other patients elective surgery was done with exploratory laparotomy in 1, segmental resection of the left colon in 2, and left hemicolectomy in 3. All surgical procedures were done in one stage. There was one postoperative death resulting from a massive pulmonary embolus, but the other 5 recovered with no further bleeding.

In our case we were confronted with the problem of massive melena that would not respond to conservative measures. The patient was in such poor condition that extensive surgical procedures seemed hazardous. The roentgenographic findings were those of diverticulosis throughout the entire colon with an area of diverticulitis in the sigmoid region. A transverse colostomy was done as an emergency procedure to control the acute hemorrhage. Five weeks later a left hemicolectomy with colosigmoidostomy was done with complete recovery.

### CASE REPORT

E. M. was admitted to the hospital on March 3, 1953, complaining of severe rectal bleeding. About six hours prior to admission he experienced a desire for a bowel movement. His stool was composed almost entirely of blood. Following this in rapid succession he had several more bowel movements of blood. There was no pain except for cramping and a bowel movement would relieve this. He fainted following a bowel movement about one hour before admission. The system review was negative. The past history was not remarkable other than having had typhoid fever during childhood. The family history did not reveal any familial diseases.

*Physical Examination:* The temperature was 97 F., pulse 100, respirations 20 and blood

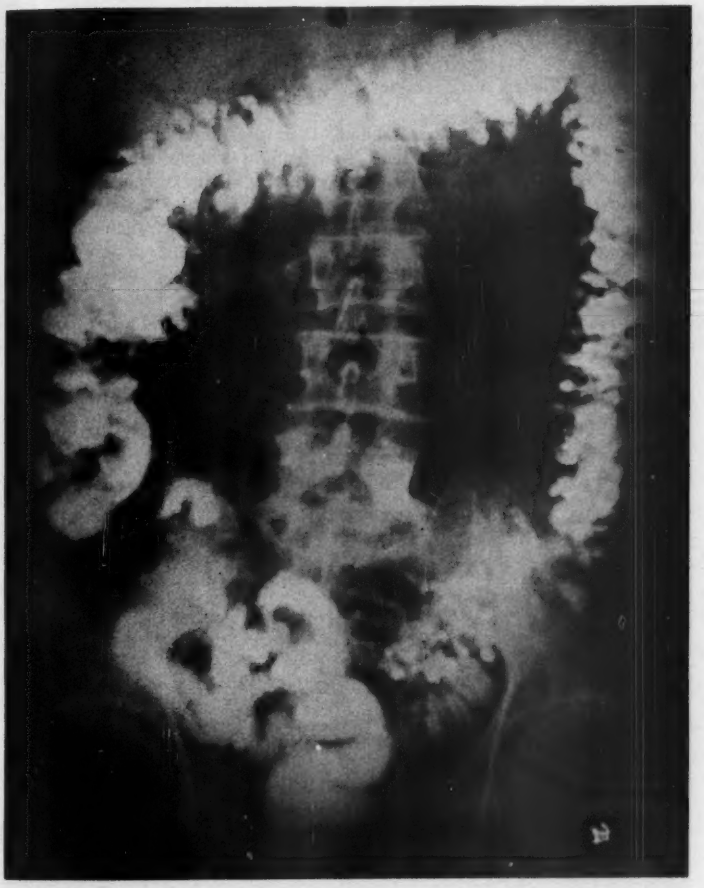

FIG. 1. Barium enema showing extensive diverticulosis of entire colon and area of diverticulitis in sigmoid region.

pressure 90/50. The skin was cool and moist. The heart rhythm was irregular. Hypospadias was present. Rectal examination revealed only the presence of a tarry stool.

*Laboratory:* The red blood cells were 3,700,000 per cu. mm., the hemoglobin was 10 Gm. per 100 cc., the white blood cells were 10,400 per cu. mm. with polymorphonuclears 64 per cent, lymphocytes 14 per cent and transitional cells 22 per cent. The urinalysis was normal except the microscopic finding of 40 to 50 white blood cells per high power field. Serologic examination was negative.

On admission he was given demerol, vitamin K, and 500 cc. of whole blood. His blood pressure rose to 105/60. Seven hours after admission a repeat blood count showed 3,450,000 red blood cells per cu. mm. and hemoglobin was 9.4 Gm. per 100 cc. He continued to improve and a barium enema was done on March 4 (fig. 1). It revealed a very extensive diverticulosis of the colon with a segment near the junction of the descending and sigmoid portions probably showing evidence of diverticulitis. The possibility of a neoplasm in this region could not be ruled out. On March 5, the patient was given an enema to help remove the retained barium. Two hours later he had another episode of severe bleeding and passed large dark red clots.

His blood pressure dropped to 80/70. He was given 1000 cc. of whole blood and his blood pressure returned to 110/70 with pulse of 80. The bleeding stopped for two days, but on March 7, the bleeding resumed with passage of copious quantities of tarry stool containing many dark red clots. Despite rapid transfusions his blood pressure dropped to 76/54 and pulse rose to 112.

He was taken to the operating room and under local anesthesia, supplemented by sodium pentothal, a transverse colostomy was done. No attempt was made to explore the peritoneal cavity due to the patient's poor condition. An obstructing clamp was placed across the colostomy loop. He received 500 cc. of blood during the procedure and left the operating table in fair condition. Following the operation he received whole blood transfusions, vitamin K, vitamin B and C, calcium gluconate, procaine penicillin G., and dihydrostreptomycin. During the next several days his blood count rose and he had no further evidence of any severe melena. The colostomy was decompressed on the second postoperative day and completely divided on the fourth postoperative day. The stool from the proximal loop did not show any evidence of blood. He continued to have some fever and the chemotherapy was changed to oxytetracycline.

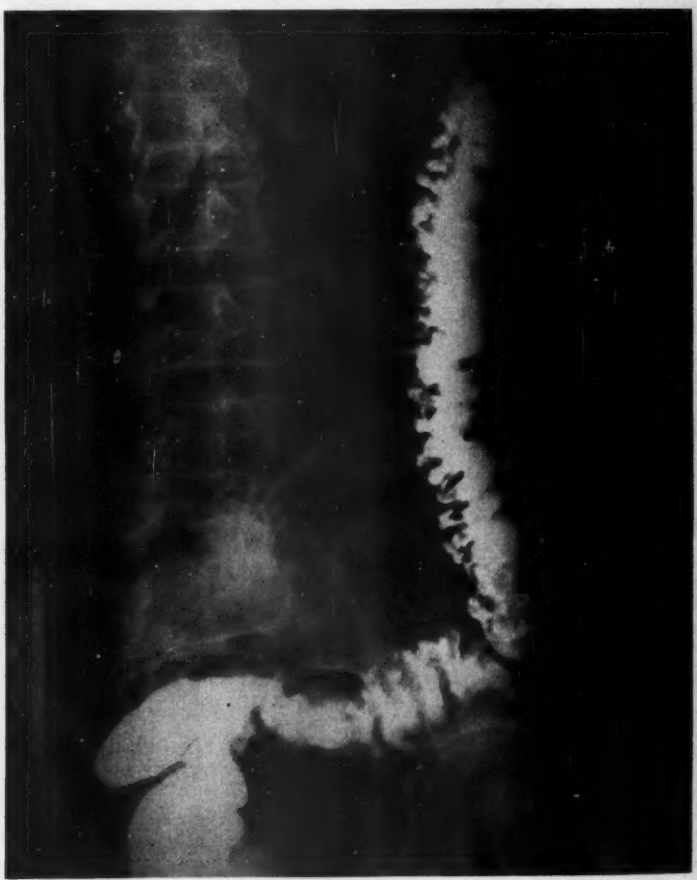

FIG. 2. Barium enema following transverse colostomy. Questionable area in sigmoid region is definitely diverticulitis rather than carcinoma.

During the next several weeks the patient improved. On March 30, the hemoglobin was 11.6 Gm. per 100 cc. He was slowly gaining weight. Irrigation of the distal side of the colostomy still gave some tarry stool return. A repeat barium enema (fig. 2) was done on March 31, and the previous findings were confirmed with no evidence of neoplasm. The study was also made of the colon proximal to the colostomy (fig. 3) and there was marked diverticulosis of the right colon. A sigmoidoscopy on April 8, was negative to 15 cm. above the anus. He was then placed on usual preparation for bowel surgery with modifications made because of the presence of the colostomy.

On April 15, a left hemicolectomy was done. The left half of the colon from the colostomy site down to the rectosigmoid region was removed. The hepatic flexure was mobilized and an anastamosis was made between the transverse colon and the lower sigmoid colon. Fifty cm. of left colon were removed. It showed numerous diverticula but no ulcerations or acute inflammatory processes. No evidence of site of hemorrhage was found. The appendix was removed and a rubber catheter was placed through the base of the appendix to decompress

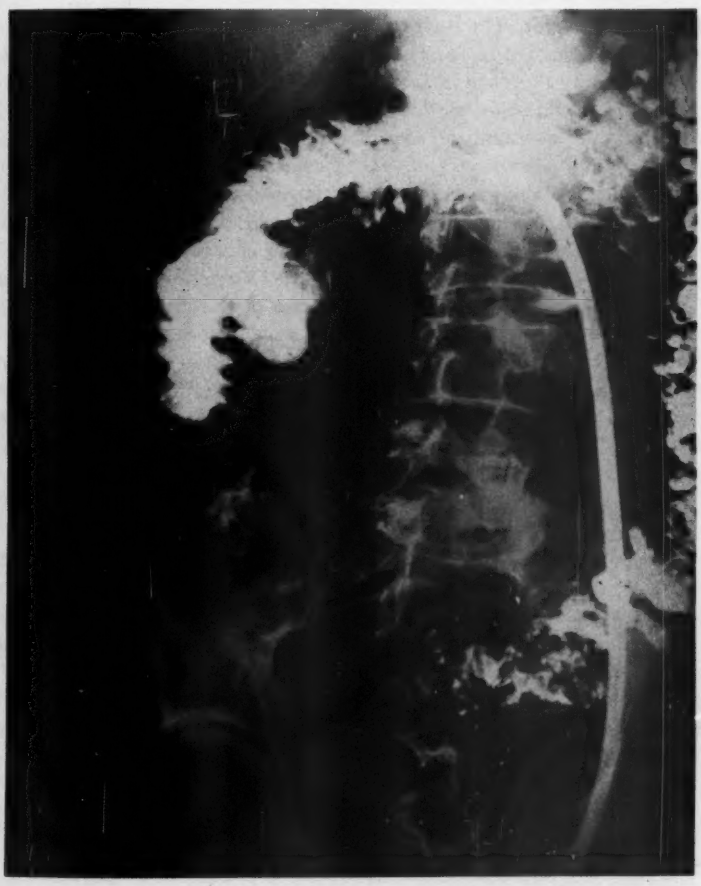

FIG. 3. Barium enema with barium filling of right colon through Foley catheter. Extensive diverticulosis of right colon is noted.

the proximal colon. On the fourth postoperative day he had a bowel movement and the cecostomy tube was removed on the tenth postoperative day. He was discharged on the fourteenth postoperative day. Since that time he has had no further bleeding, is gaining weight and feels good. He remains on a low residue diet.

## COMMENT

The barium enema study on March 4, 1953, suggested the sigmoid region as being the most likely source of hemorrhage. When the transverse colostomy was done the patient was in very poor condition and it was doubtful if he could have withstood any major exploration or resection. Apparently by diverting the fecal stream the local trauma of feces to the area of hemorrhage was stopped and a clot could form.

Even though the subsequent barium enema on March 31, 1953, revealed extensive diverticulosis throughout the entire colon it was decided to resect only the left half of the colon. The reason for this decision was the cessation of hemorrhage following diversion of the fecal stream in the midtransverse colon region and absence of any evidence of bleeding from the right half of the colon. The lower level for resection in the lower sigmoid region was determined by both sigmoidoscopy and examination of this region of the colon at the time of the resection.

It is possible that this patient will develop a diverticulitis in the remaining diverticula as the fecal stream becomes more solid. Conservative medical management should control this and the low incidence of severe melena in diverticulitis makes it unlikely that he will require further surgical intervention. With so much of his colon remaining he should be free of the intestinal complications sometimes seen following total colectomy.

The complete lack of symptoms prior to the acute onset of the massive melena seemed somewhat unusual. We were not surprised that a careful examination of the resected portion of the colon failed to reveal the site of the hemorrhage. Dr. Cate[1] could not demonstrate the exact site of the hemorrhage, even though the resection was done in his case during the period of acute hemorrhage.

## SUMMARY

A case report of massive melena secondary to diverticulitis of the sigmoid and extensive diverticulosis of the entire colon is reported.

A transverse colostomy was done as an emergency procedure to control the acute hemorrhage. A left hemicolectomy was done five weeks later with anastomosis of the transverse colon to the lower sigmoid colon. This patient has been free of symptoms following recovery from the second operation.

In poor-risk patients this method of treatment may be life saving and allow definitive surgery to be done at a later date.

## REFERENCES

1. Cate, W. R., Jr.: Colectomy in treatment of massive melena secondary to diverticulosis, Ann. Surg. *137:* 588 (April) 1953.
2. Quinn, W. C., and Ochsner, A.: Bleeding as complication of diverticulosis or diverticulitis of colon, Am. Surgeon *19:* 397 (May) 1953.
3. Stone, H. B.: Large melena of obscure origin, Ann. Surg. *120:* 582 (Oct.) 1944.

# MASSIVE SPLENIC INFARCTION WITH ACUTE NECROSIS AND ABSCESS FOLLOWING LIGATION OF THE HEPATIC AND SPLENIC ARTERIES FOR PORTAL HYPERTENSION

## CASE REPORT

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Ligation of the splenic artery without removal of the spleen has been used in the management of selected patients with various lesions of the spleen. Until recently, its use has been reserved principally for poor-risk patients in whom indicated splenectomy would be too hazardous or impossible without causing death. Under such circumstances it has been used for the management of Banti's syndrome, portal hypertension with splenomegaly, and thrombocytopenic purpura.

This procedure was first successfully performed by Blaine[5] in 1913 upon a patient who had Banti's syndrome and whose condition was critical. Because of firm adherence of the enlarged spleen posteriorly and superiorly by vascular adhesions, splenectomy was considered too hazardous and the splenic artery was ligated 3 cm. from the spleen. Except for a febrile course during the first two postoperative days, the patient made an uneventful recovery until approximately four and a half weeks later when a painful bulge was noted in the center of the wound. This was opened with a hemostat with the release of a large quantity of broken down splenic tissue.

Since Blaine's report describing one ligation of the splenic artery for Banti's syndrome, Allessandri,[1] Watson,[10] Berg and Rosenthal,[2] Everson and Cole,[6] and Linton and Hardy[7] have reported their experiences with this procedure. All have recommended its use in selected cases, and none has observed deleterious effects attributable to rapid splenic necrosis. Experimental studies described by Blaine in his original report substantiated his clinical observations. His experimental data indicated that following splenic artery ligation immediate shrinkage of the splenic pulp occurred in dogs. Furthermore, he reported that all of the dogs so treated survived the procedure. At second operations, the spleens were noted to be atrophied and wrapped in omentum.

During the past three years, ligation of the splenic artery along with the hepatic artery has been recommended by Berman,[3] and Rienhoff[9] in patients other than those who are poor risks as a method of reducing portal hypertension in the presence of recurrent bleeding from esophageal varices or recurrent ascites. Removal of the spleen during this procedure has not been recommended in the belief that slow atrophy of this organ would follow rather than acute necrosis or abscess formation.

Within the past three years, 18 patients who had cirrhosis with portal hyper-

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tension have had ligations of the splenic and hepatic arteries by members of the Surgical Department of the Cincinnati General Hospital and University of Cincinnati. In 1 of the patients operated upon by us, acute necrosis of the spleen occurred, but was not recognized before autopsy. Since the death was directly attributable to ligation of the splenic artery, the following case report is presented.

## CASE REPORT

J. S., a 56 year old white man, was a shipping clerk who was admitted to the Medical Service of the Cincinnati General Hospital on Aug. 9, 1951. Two weeks prior to admission he first noted tarry stools which were followed by hematemesis. His history indicated that he had imbibed at least one-half pint of whiskey daily for 15 years. Physical examination revealed a chronically ill white man who was in no distress. His blood pressure was 125/80 and his pulse was 72. The pertinent physical findings consisted of innumerable spider telangiectases over the upper body, dilated abdominal veins, ascites, a nodular liver 3 fingerbreadths below the costal margin, and protuberant hemorrhoids.

Laboratory work revealed red blood cells of 3.83 million and hemoglobin of 10.5 Gm. His liver profile was as follows: prothrombin time, 75 per cent; bromsulfalein retention, 36.5 per cent; thymol turbidity, 10 units; zinc sulfate turbidity, 23.5 units; cephalin flocculation, 3+ in 24 hrs.; total lipids, 550 mg. per cent; serum bilirubin, prompt, 1.75 mg. per cent; total, 4.3 mg. per cent; alkaline phosphatase, 4.3 units per cent; total protein, 6.30 Gm. per cent; albumin, 2.9 Gm. per cent; and globulin, 3.40 Gm. per cent.

Esophageal varices were demonstrated roentgenographically.

Under intensive medical management his cirrhosis became compensated and his bleeding stopped. He was therefore discharged on September 13.

On October 5, he was readmitted to the Medical Service with a recurrence of tarry stools and intermittent episodes of hematemesis. His hemoglobin at this time was 9.0 Gm. and his red blood cells were 2.96 million. During the first 48 hours of his second admission he was given 3000 cc. of whole blood and received 16 hours of esophageal tamponade with a Sengstoken tube. His esophageal bleeding was controlled in this manner.

After an interval of two weeks he was transferred to the Surgical Service. His preoperative chemotherapy consisted of 100,000 units of aqueous penicillin every 8 hours and 0.5 Gm. of streptomycin every 12 hours for an interval of five days prior to surgery. Ligations of both hepatic and splenic arteries and liver biopsy were performed on October 22. The liver was small, being approximately one-half its normal size, hard and diffusely nodular. The portal venous pressure was found to be 275 mm. of water. Approximately 1000 cc. of ascitic fluid was removed from the peritoneal cavity. Each vessel was individually isolated and ligated, the splenic 2 cm. and the hepatic 4 cm. from the sites of origin at the celiac axis. Following the completion of the operative procedure, a specimen of liver was removed for biopsy. The patient tolerated the procedure well and his immediate postoperative course was uneventful.

The microscopic findings of the liver biopsy revealed considerable periportal fibrosis, marked distortion of lobular architecture, variation in size of nodules, and a marked degree of bile duct proliferation in the portal connective tissues. A moderately severe chronic inflammatory reaction was noted in these areas. A microscopic diagnosis of postnecrotic cirrhosis of the liver was made from these findings.

Two weeks after operation the ascites recurred and it was again necessary to perform paracenteses once or twice weekly. The condition of the patient gradually became worse. He developed deepening jaundice and an unexplained fluctuating fever despite antibiotic therapy consisting of 100,000 units of aqueous penicillin G intramuscularly every eight hours, and 500 mg. streptomycin every 12 hours. Blood cultures during this septic course were all sterile. It was thought that necrosis or infection of the liver had occurred and the

antibiotic treatment was changed. Chloromycetin in doses of 500 mg. every six hours, and terramycin in the same dose were tried in succession. Aureomycin was then given in doses of 500 mg. intravenously every 12 hours but to no avail. His course was one of progressive deterioration and he died eight weeks postoperatively.

At autopsy, a large abscess cavity was found in the left upper quadrant. It contained approximately 600 cc. of blood-stained purulent material, culture of which was negative. What initially appeared to be a pyogenic membrane was noted to be the capsule of the spleen. The capsule was adherent to the liver anteriorly and to the peritoneum posteriorly and laterally. When the organ was removed it was found to weigh 780 Gm. The lower one-half of the spleen was soft with pericapsular fibrosis and on section the splenic pulp was purplish-red and appeared viable (fig. 1). A long tongue of similar deep red splenic tissue extended upward along the lateral surface. The entire upper pole of the spleen, however, had become liquefied (fig. 2) except for fragments of pale, tannish-pink, infarcted splenic pulp varying from 5 to 15 cm. in diameter. The splenic artery was noted to be ligated 2 cm. from its site of origin at the celiac axis. Microscopic examination of the spleen showed complete infarction with numerous scattered areas of bacteria.

The capsule of the liver was slightly thickened and adherent. The liver was pinkish-yellowish tan and grossly nodular in appearance. The nodules varied in size from 4 to 12 mm. and a few areas of gross scarring were noted. The ligature on the hepatic artery was noted to be 4 cm. from the site of origin and the vessel was completely occluded at this point. The hepatic vein was patent throughout its entire course. No areas of frank liver necrosis were noted. Microscopic examination of the liver revealed broad, irregular, fibrous bands of tissue streaming out from the portal areas, pinching off nodules of liver parenchyma. There was bile duct proliferation and much inflammatory exudate was noted. Liver cell necrosis was striking and a few vessels contained purulent exudate. The picture represented portal cirrhosis of the mixed type.

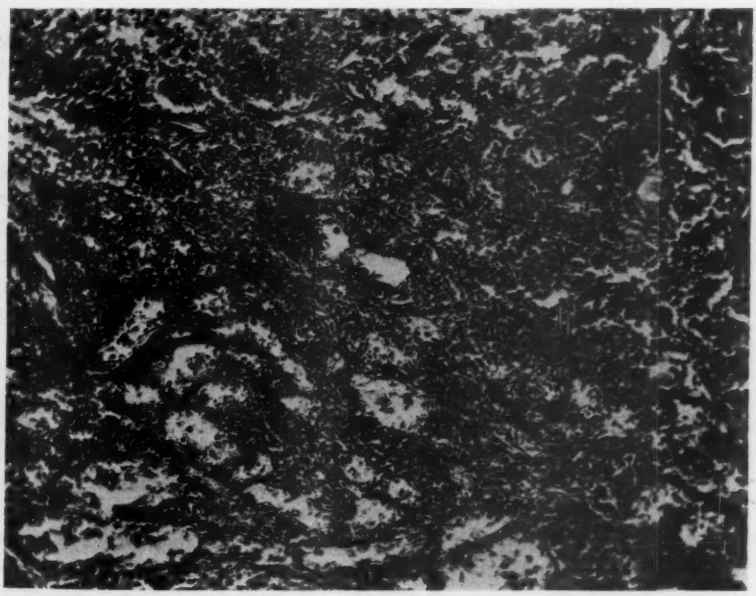

FIG. 1. Section from lower pole of spleen showing viable splenic pulp

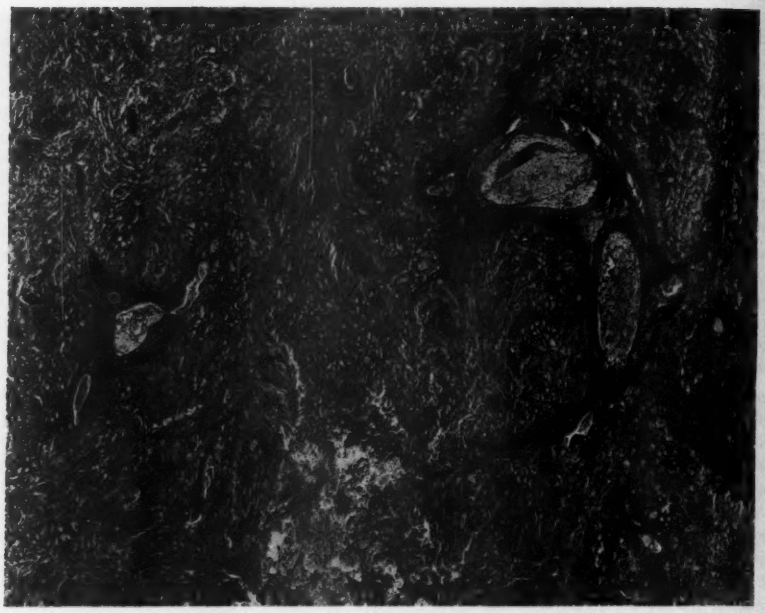

FIG. 2. Section from upper pole of spleen showing complete infarction

The immediate cause of death was attributed to massive splenic infarction with suppuration.

## DISCUSSION

Ligation of the hepatic artery in the treatment of portal hypertension is associated with certain known hazards. Necrosis of the liver, hepatic vein thrombosis, decrease in total liver blood flow, and liver failure have all been noted. Rienhoff[9] and Berman and Fields[4] have recently shown that these can be controlled generally by the careful selection of the patients, proper use of antibiotics, and the surgical procedures described by them. The development of hepatic abscesses following ligation of the hepatic artery can be prevented by judicious use of antibiotics.[8]

Ligation of the splenic artery, however, has been thought to be a safe procedure. The general belief that gradually atrophy of the spleen always occurs after ligation of the splenic artery resulted in our overlooking the spleen as a possible source of pathology and explanation for this patient's complicated postoperative course. His sepsis associated with deepening jaundice was thought to be the result of intrahepatic necrosis and abscesses following ligation of the hepatic artery, despite the intensive chemotherapy administered. The diagnosis might have been established by a more intensive diagnostic work-up including fluoroscopy of the diaphragms, serial roentgenograms of the chest and a barium enema.

In retrospect, the small shrunken liver and poor condition of this patient gave

him a poor prognosis with any form of treatment. However, if incision and drainage of the abscess had been done, his toxic and septic course might have been significantly altered.

## CONCLUSIONS

Since ligation of the splenic and hepatic arteries is being used in the management of a limited but specific group of patients with portal hypertension and various splenic lesions, the inherent hazards of this procedure have been discussed.

In addition to the already known hazards associated with hepatic artery ligation, namely necrosis of the liver, hepatic vein thrombosis, decrease in total liver blood flow, liver failure, and intrahepatic abscesses, an additional complication of massive splenic infarction with acute necrosis and abscess formation encountered in one of our patients has been presented.

## REFERENCES

1. Alessandri, R.: Experiences with surgery of spleen: report of two unusual cases, J. Mt. Sinai Hosp. *4:* 489 (March–April) 1938.
2. Berg, A. A., and Rosenthal, N.: Ligation of splenic artery for thrombocytopenic purpura and congestive splenomegaly, J. Mt. Sinai Hosp. *8:* 382 (Jan.–Feb.) 1942.
3. Berman, J. K.; Koenig, H., and Muller, L. P.: Ligation of hepatic and splenic arteries in treatment of portal hypertension; ligation in atrophic cirrhosis of liver, A.M.A. Arch. Surg. *63:* 379 (Sept.) 1951.
4. Berman, J. K., and Fields, D. C.: Present Status of Hepatic, Splenic and Left Gastric Arterial Occlusions in Advanced Portal Cirrhosis of Liver. Presented at the meeting of the Western Surgical Association, December 1953.
5. Blain, A. W.: Ligation of splenic artery, Surg., Gynec. & Obst. *26:* 660 (June) 1918.
6. Everson, T. C., and Cole, W. H.: Ligation of splenic artery in patients with portal hypertension, Arch. Surg. *56:* 153 (Feb.) 1948.
7. Linton, R. R., and Hardy, I. B., Jr.: Surgery of portal hypertension; portacaval shunts and two-stage method in poor-risk patient, J. Michigan M. Soc. *48:* 1005 (Aug.) 1949.
8. Markowitz, J.; Rappaport, A., and Scott, A. C.: Prevention of liver necrosis following ligation of hepatic artery, Proc. Soc. Exper. Biol. & Med. *70:* 305 (Feb.) 1949.
9. Rienhoff, W. F., Jr.: Ligation of hepatic and splenic arteries in treatment of portal hypertension with report of six cases; preliminary report, Bull. Johns Jopkins Hosp. *88:* 368 (April) 1951.
10. Watson, R. B.: Ligation of splenic artery for advanced splenic anemia, Brit. M. J. *1:* 821 (April 20) 1935.

# HYPERTENSION AND UNILATERAL RENAL DISEASE

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The relationship between unilateral renal disease and hypertension is one of the most controversial problems in medicine. It is accepted that diseases of the renal parenchyma such as glomerulonephritis, nephrosclerosis, and chronic pyelonephritis, are frequently accompanied by hypertension even before diminution in renal function is demonstrated. In polycystic kidney disease approximately 65 per cent of the patients develop hypertension. Prior to the present concept of essential hypertension there was widespread belief that chronically elevated blood pressure never existed in the absence of renal disease. This concept was later changed to the belief that renal disease and hypertension have no relationship. The present belief is that they are related in specific instances. One of these specific instances, unilateral renal disease, is the basis for this paper.

Butler[3], in 1937, was the first clinician to report a case of an individual with elevated blood pressure in which unilateral renal disease was demonstrated and removal of the diseased kidney resulted in a cure. In 1880, Levinsky[8] produced cardiac hypertrophy within a few weeks time by constriction of the renal artery. In 1898 Tigerstedt and Bergman[11] introduced the possibility of an intarrenal humoral mechanism in hypertension by injecting saline extract of normal kidney tissue into experimental animals. They isolated renin. In 1905 Kagtenstein[7] produced transitory hypertension by loosely ligating the renal pedicles of experimental animals. In 1909 Alwens[1] achieved a slight hypertension by compressing the kidney in an onkometer. In 1929 Hortwich[6] (Vollard) noted hypertension following bilateral ligation of the ureters and following constriction of the renal artery with small silver rings. In 1933 the first report of Goldblatts[4, 5] work directed clinicians attention to unilateral renal disease as a cause for hypertension.

## CLINICAL DATA

The exact etiology of renal hypertension is not known in spite of the volume of experimental work which has been reported. It is believed that the basic substance is renin, which is not hypertensive in itself but is thought to combine with hypertensinogen to produce one or more compounds, hypertensin. These produce constriction and subsequent elevation in blood pressure.

According to Brassch[2] there are two hypotheses; (1) the antipressor hypothesis, whereby the kidney is thought to fail to detoxify a normally existing pressor compound, and (2) that hypertension occurs because the kidney fails to produce its' normal supply of humoral agent. Also as pointed out by Brassch, no specific

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Presented at the annual meeting of the Southwestern Surgical Congress in Salt Lake City, Utah, Sept. 23, 1953.

pathologic renal lesion is found which is consistently associated with hypertension. However, the lesions most commonly associated with unilateral renal disease causing hypertension are specific or nonspecific pyelonephritis as seen primarily or secondarily superimposed upon hydronephris or nephrolithiasis. The severity of the parenchymal atrophy and vascular sclerosis in the above lesions is in direct proportion to the incidence of hypertension.

From the literature it is evident that nephrectomy has been done for every type of renal lesion with the hope of relieving hypertension. The fact that a specific lesion is not always related to hypertension makes it difficult to select these patients for surgery. Also there is no diagnostic test which will indicate the outcome following surgical removal of the diseased kidney. As pointed out by Sabin,[10] the diseased kidney should be demonstrated to have markedly impaired function before operation is contemplated. The opposite kidney should be normal functionally, bateriologically and morphologically. Also the best clinical results are obtained on the younger patient. The duration of the hypertension is another factor of importance, since hypertension of long standing, with irreversible arteriosclerotic changes in the good kidney, may render the chance of a cure by nephrectomy small indeed.

## CASE REPORT

Mrs. D. O., aged 28, was admitted to St. Mark's Hospital on the service of one of us (J. C.) for a diagnostic workup. She gave a history of the onset of hypertension five years

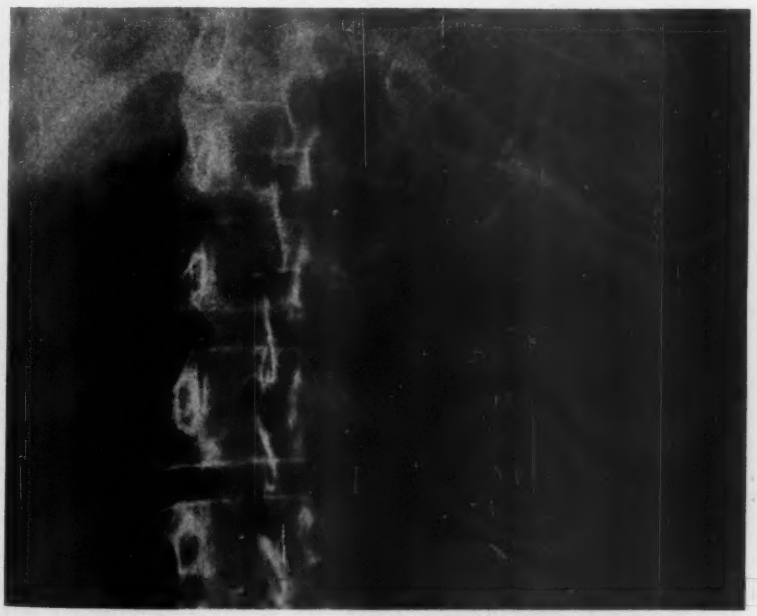


FIG. 1. Flat Plate of Abdomen showing calcification of left kidney

prior in the eighth month of pregnancy. Labor was induced and she delivered a normal baby following which her blood pressure returned to normal six weeks postpartum. Three months ago labor had to be induced because of severe hypertension of 230/140. At this time she was advised against future pregnancies. She had no menstrual periods since last delivery. The patient's mother gave a further history of an accident to the patient at the age of 5 years. At that time the left side of the patient's body was injured by the wheel of a wagon passing over it. She passed gross hematuria and was confined to bed for a week. There was no further history of urinary symptoms or difficulty up to the present admission.

On physical examination she was found to be well nourished and well developed. Her blood pressure was 160/114. Fundi showed Grade II narrowing of the arteries. Her heart border was in normal position but there was a Grade II systolic murmur audible in the eleventh interspace. Pulsations were normal in the peripheral vessels. No abnormal masses were palpable. A urologic examination was negative.

The urinalysis showed a specific gravity of 1.028, a trace of albumin, no sugar, 3 to 5 leukocytes and 5 to 10 erythrocytes per high power field. The hemoglobin was 72 per cent, red blood count 4,200,000 per cu. mm. and white blood count 8,850 per cu. mm. The blood urea nitrogen was 14.7. The phenolsulfonphthalein function was 73 per cent total in 60 minutes. A pregnancy test was positive.

An obstetrician suggested that, in view of the history of hypertension for the past five years with toxemia of pregnancy, the pregnancy (14 weeks) be terminated by hysterotomy followed by sterilization. His suggestion was not accepted.

The patient was evaluated medically by an internist who suggested that an excretory urogram be done prior to any type of surgery. A urogram showed a large calcified hydronephrotic left kidney with reduced function and a normal (compensatory hypertrophy) right kidney (figs. 1 and 2). A cystoscopic examination was made. A no. 20 F. cystoscope was

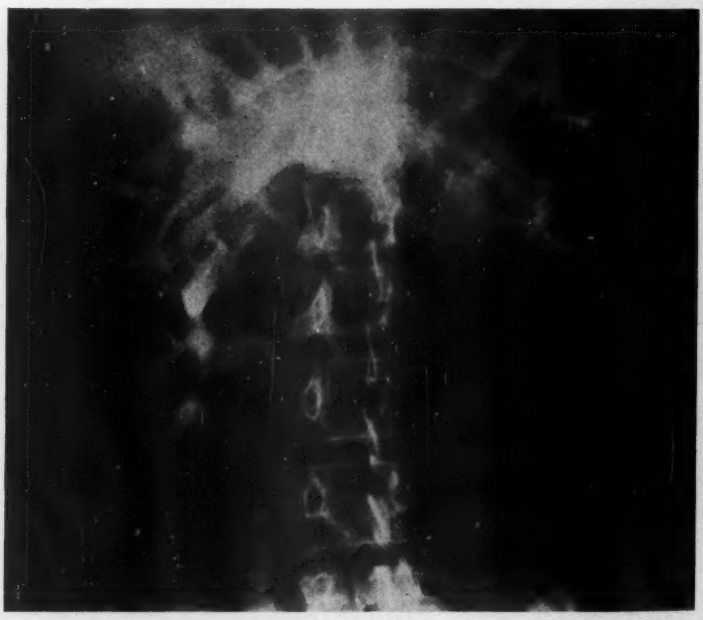


FIG. 2. Intravenous Urogram showing normal right kidney with compensatory hypertrophy and calcified Left Kidney.

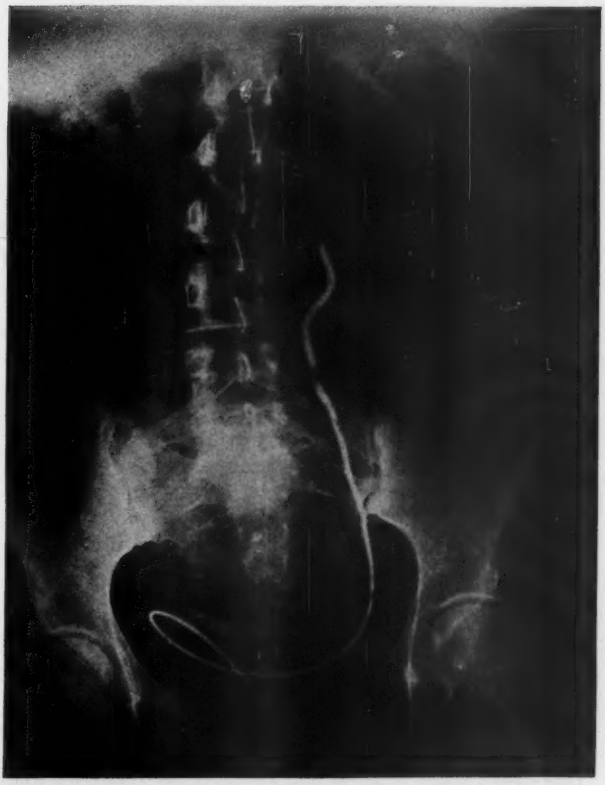


FIG. 3. Attempted Left retrograde study meeting complete obstruction in upper third of ureter.

introduced without difficulty. The urethra and bladder appeared to be normal. The spurts of urine from the right ureteral orifice came at regular intervals. None was seen coming from the left. A no. 5 catheter met obstruction corresponding to a point in the middle third of the ureter. This could not be bypassed. Dye was injected with the patient in the Trendelenburg position. Dye did not bypass the point of obstruction and it was impossible to outline the kidney pelvis (figs. 3 and 4).

The diagnosis of hypertension due to unilateral renal disease was agreed upon and a left nephrectomy was done on March 25, 1948. The left kidney was explored through a lumbar incision. Great difficulty was encountered in trying to free the renal mass from surrounding structures. There was considerable calcification in the renal parenchyma and the hydronephrotic sac contained urine. The patient tolerated the operation well.

### PATHOLOGISTS REPORT

*Description of gross specimen:* "The specimen is an enlarged kidney, almost totally replaced by a large hydronephrotic sac measuring 24 by 20 by 18 cm. The dilated calyces vary from 2.5 to 10 cm. in diameter surrounded by a very thin layer of renal parenchyma. No ureter is attached. Much of the wall is calcified, and the mass shows adhesions to fatty tissue and even muscle in places."

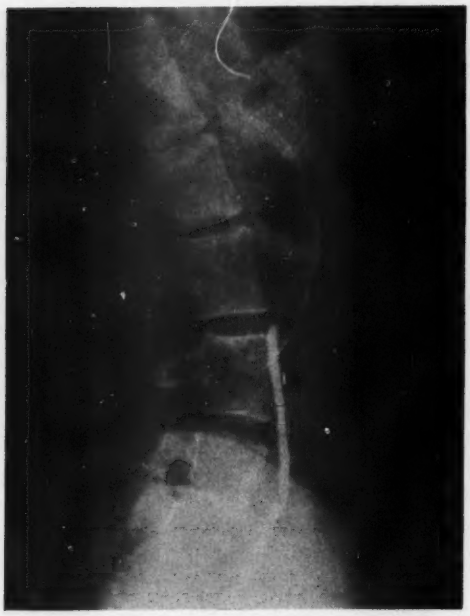
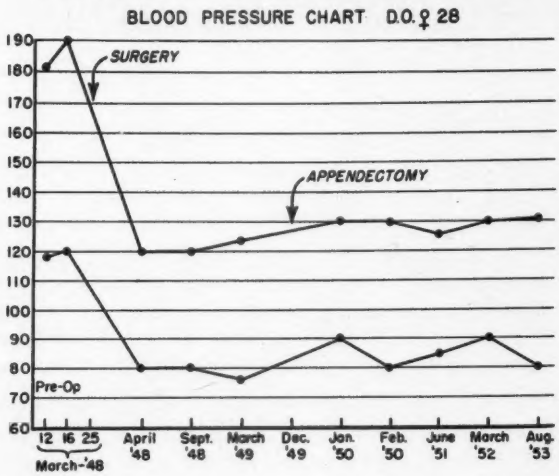


FIG. 4. Lateral view of figure 3

CHART I

*Blood pressure excursion preoperative and postoperative*



"Microscopic examination shows mildly and focally edematous connective tissue that exhibits calcification, and shows attached connective tissue with a little muscle tissue on its outside. Well formed glomeruli are scarce and there are 7 or 8 distorted structures that are cystic, stained by a bluish material that probably represent greatly distorted renal tubules."

The patient's blood pressure fell to an average level of 128/80 postoperatively, with a range of from 132/80 to 126/85 (Chart I). Her course was uneventful and she was discharged from the Hospital on the tenth postoperative day. Her pressure remained normal throughout the following six months of pregnancy and she delivered a normal term baby boy without difficulty. Two years later one of us (J. C.) removed her appendix.

## DISCUSSION

The fact that a specific lesion is not always present to cause hypertension in unilateral renal disease makes the diagnosis and prognosis difficult to evaluate. The most common lesion associated with this entity is chronic pyelonephritis into which class the reported case falls. A few facts are definitely evident on close perusal of the literature. They are: 1. Age of the patient. Best results are often obtained in the young patient, i.e., below 45 years of age. 2. Duration of hypertension. Patients with symptoms of two years duration or less have the best outlook. 3. Condition of the kidney. Reduced function and/or atrophy of the diseased kidney with evidence of compensatory hypertrophy in the healthy kidney offer best results. 4. Careful selection of the patient for nephrectomy is essential for a good result. Even though only 2 per cent of all essential hypertensive patients will be found to have surgical renal disease they are entitled to a urologic survey. This may consist of a flat plate of the abdomen, followed by an excretory urogram and then cystoscopy and retrograde study if indicated. Pappel and Alyea,[9] thought that, in a carefully selected group of patients with essential hypertension in association with unilateral surgical renal disease, one can expect about 34 per cent to return to permanent normatension following nephrectomy. 5. Claim of cure should be made only after prolonged follow-up and careful evaluation.

## SUMMARY

A sketch of the experimental work concerning the renal origin of hypertension is presented. The fact that the etiology of renal hypertension is not yet known is stressed.

The case report of a patient who had a nephrectomy during pregnancy for renal hypertension, followed by delivery of a normal baby without recurrence of hypertension up to the present time (over five years) is presented.

## REFERENCES

1. Alwens, quoted by Boeminghaus, H., and Götzen, F. J.: Über den Hochdruck bei einseitiger Nierenerkrankung; Experiment, Kasuistik und praktische Folgerungen, Zschr. Urol. *45:* 472, 1952.
2. Braasch, W. F.: End results following nephrectomy in patients with hypertension, J. Urol. *68:* 6 (July) 1952.
3. Butler, A. M.: Chronic pyelonephritis and arterial hypertension, J. Clin. Investigation *16:* 889 (Nov.) 1937.

4. Goldblatt, H.; Gross, J., and Hanzal, R. F.: Studies on experimental hypertension, effect of resection of splanchnic nerves on experimental renal hypertension, J. Exper. Med. *65:* 233 (Feb.) 1937.
5. Goldblatt, H.: The Renal Origin of Hypertension, Springfield, Ill., Charles C. Thomas, 1948.
6. Hartwich, A.: Der Blutdruck bei experimenteller Urämie und partieller Nierenausscheidung, Ztschr. f. d. ges. exper. Med. *69:* 462, 1930.
7. Katgenstein, quoted by Boeminghaus, H., and Götzen, F. J.[1]
8. Levinsky, quoted by Boeminghaus, H., and Götzen, F. J.[1]
9. Puppel, A. D., and Alyea, E. P.: Hypertension and surgical kidney, J. Urol. *67:* 433 (April) 1952.
10. Sabin, H. S.: Hypertension in unilateral renal disease, J. Urol. *59:* 8 (Jan.) 1948.
11. Tiegerstedt, R., and Bergmann, P. G.: Niere and Kreislauf, Skandinav. Arch. f. Physiol. *8:* 223 (Nov.) 1898.

# THE TREATMENT OF CHRONIC ARTHRITIS WITH A COMBINATION OF COBRA VENOM, FORMIC ACID, AND SILICIC ACID

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The purpose of this article is to report a new preparation for the treatment of chronic arthritis. The medication is a combination of cobra venom with formic and silicic acids. A great timidity is felt in offering another treatment for arthritis to the profession, but the confidence which several clinicians have in its merit gives encouragement, as does the enthusiasm of patients who have benefited from it. Of the 466 patients whose treatment began before Sept. 1, 1952, 91.4 per cent achieved satisfactory results. A much larger series is under study and will be reported at a later date.

This report is divided into two main parts. The first is taken from the case records and deals with the type of arthritis, the severity, and the results as seen by the physician (table I). In this section results are entered as either satisfactory or unsatisfactory after considering all the evident factors—physical and mental status, extent and duration of joint and muscle injury. The second part summarizes the patients' ideas of their conditions before, during, and after treatment (table II). The information in this section was of course secured by questioning the patients (277). Those patients who still are under treatment were allowed three alternatives as to their progress: (1) unsatisfactory, (2) encouraging, (3) satisfactory. Those who had finished treatment were allowed only two choices: satisfactory or unsatisfactory.

## ADMINISTRATION

The solution, containing cobra venom, formic acid and silicic acid,* is a clear, water-white liquid, quite stable for at least a year under ordinary refrigeration. It is injected with a 25 or 26 gauge needle subcutaneously close under the skin, usually in the deltoid region or in the anterior part of the thighs 3 or 4 inches above the knees. The injection should produce a perceptible elevation. This insures infiltration of the deep layers of the skin, which seems essential for the maximum effect. Treatments are given at weekly intervals. The usual sequence of dosage is 1 cc., 1.5 cc., 2 cc., 2.5 cc., and 3 cc. The injection is divided into two equal parts for simultaneous injection into different sites. If given slowly, the stinging effect is negligible. In patients who are hypersensitive to pain, 0.5 cc. of procaine 2 per cent may be added to each dose in the syringe. The administration of the larger quantities often requires a full minute to avoid discomfort. Usually the quantity given can be increased as suggested, but this should not be done if a large tender lump from the previous treatment is present at the end of a week. The interval between treatments at the start is one week, increased to two weeks

*"NYLOXIN," Hynson, Westcott & Dunning.

TABLE I

*Clinical evaluation of 466 unselected, consecutive cases*

Treated from 1944 to Sept. 1, 1952

| No. | By Type | | | By Severity | | | By Results | |
|---|---|---|---|---|---|---|---|---|
| | Osteo | Rheum. | Mixed | Mild | Average | Severe | Satisfactory | Unsatisfactory |
| 466 | 344 | 74 | 48 | 31 | 276 | 159 | 426 | 40 |
| % | 74% | 16% | 10% | 7% | 59% | 34% | 91.4% | 8.6% |

TABLE II

A. Patients Estimate of Treatment; 277 Patients Interviewed

1. Patients Estimate of Pain

a. Before Treatment

| No. | Mild | Moderate | Severe |
|---|---|---|---|
| 277 | 16 | 103 | 158 |
| % | 6% | 37% | 57% |

b. After Treatment

| No. | Unchanged | Improved | Absent |
|---|---|---|---|
| 277 | 12 | 160 | 105 |
| % | 5% | 57% | 38% |

2. Patients Estimate of Disability

a. Before Treatment

| No. | Slight | Moderate | Severe |
|---|---|---|---|
| 277 | 84 | 110 | 83 |
| % | 30% | 40% | 30% |

b. After Treatment

| No. | Unchanged | Improved | Restored to Normal |
|---|---|---|---|
| 277 | 19 | 152 | 106 |
| % | 7% | 55% | 38% |

3. Patients Reporting Swelling at Start: (216)

| No. | Unchanged | Improved | Absent |
|---|---|---|---|
| 216 | 13 | 100 | 103 |
| % | 6% | 46% | 48% |

4. Patients Reporting Limited Flexibility: (256)

| No. | Unchanged | Improved | Normal |
|---|---|---|---|
| 256 | 20 | 158 | 78 |
| % | 8% | 62% | 30% |

B. Patients Under Treatment since Sept. 1, 1952: (176)

1. Patients Estimate of Progress

| No. | Unsatisfactory | Encouraging | Satisfactory |
|---|---|---|---|
| 176 | 0 | 41 | 135 |
| % | 0% | 18% | 82% |

C. Patients Reporting on Status after Cessation of Treatment (6 months to 5 years): (94)

| No. | Unsatisfactory | Satisfactory |
|---|---|---|
| 94 | 6 | 88 |
| % | 6.4% | 93.6% |

Note: No patient has reported any unfavorable result upon health, concomitant disease, nor has there been a single report of exacerbation of the arthritis.

when full control of symptoms is established. Usually after 5 to 10 treatments two weeks apart the interval can be lengthened to 3 or 4 weeks, and gradually by increments of 2 weeks to once every 3 months, at which interval most patients elect to stay, for fear of a relapse. However, many have been able to stop treatment entirely without recurrence of symptoms for periods up to five years. It was found that shallow injection involving the skin augmented the effect of the drug, increasing the number of favorable responses and still further shortening the average improvement time.

### MANNER OF DEVELOPMENT

In the search for a long-acting, non-narcotic, pain-relieving agent, experiences were remembered with cobra venom in cases of terminal malignant disease. In September 1944, this agent was tried out on arthritic patients, with definite improvement in the results. In 1945, after reading of some promising experiments with the injection of silicic acid into the vicinity of arthritic joints, the author attempted to prepare a cobra venom silicic acid solution with formic acid as a buffer. With the research department of Hynson, Westcott & Dunning, Inc., a stable preparation has been made. This product has been tested in the arthritis clinics of the Charity Hospital in Cleveland and the Maryland General Hospital in Baltimore. The results of the study in Baltimore are being reported by Lumpkin and Firor. Over 4,000 injections have been given in the past six months to 150 patients. The clinical results in these patients have been the same as in the author's experience. The results with the combination of the three substances have been far superior to those obtained when the materials were given separately.

### GENERAL DESCRIPTION OF THE 466 CASES IN THIS SERIES

The group from which these data are taken numbers 466 completely unselected consecutive cases, treated before Sept. 1, 1952. During this time roughly 150 other patients started treatment but did not continue, receiving only from one to six injections, so were regarded as of no statistical value since the average time for initial improvement is from six to eight weeks. The age range of the 466 patients was from 18 to 95 years, and the duration of symptoms from 2 months to 40 years.

Some exactness of diagnosis has been sacrificed for simplicity of classification; thus only osteoarthritis, rheumatoid, and mixed types are given, and variants assigned to the closest general type—hence Marie-Struempel and Post-Still's disease are included with the rheumatoid group. Severity is gauged as mild, average, or severe. Results in this section are tabulated simply as satisfactory or unsatisfactory.

### DISCUSSION OF TABULATION RESULTS

It is found that almost invariably the patient gauges the severity of his disease by the degree of pain he suffers. However, of the 57 per cent reporting their pain as severe, only 34 per cent appeared to the physician to warrant that term.

In table II, part B: "Patients under treatment since Sept. 1, 1952; Patients Estimate of Progress", the astonishing 100 per cent total of encouraging or satisfactory results were considered as springing from the betterment of general health and the lessening of emotional tension in relation to their disability, rather than an exact statement of the facts. Perhaps the most interesting report is that of the group of 94 patients reporting 6 months to 5 years after cessation of treatment. Of these, 93.6 per cent state that their status has remained satisfactory. These patients, together with a large number who continue treatment at 2 to 4 month intervals although symptom free, are the basis of the claim that clinical arrest of the disease is possible in a high percentage of patients. To corroborate the patients' impression that the disease is arrested, the author has in some cases conclusive roentgenographic evidence.

### EFFECTS ON SYMPTOMS OFTEN CONNECTED WITH ARTHRITIS

1. *Improvement in General Health.* All but 2 of the 466 patients stated that their general health had improved. This improvement begins usually in from 4 to 6 weeks. It consists of lessening of the mental depression, better appetite, increased strength, or lessening of the feeling of heaviness in the extremities. This improvement often is accompanied with rises in hemoglobin and red cell count levels.

2. *Lowering of Abnormal Blood Pressure.* Of the 466 patients, 268 had systolic pressures ranging from 140 mm. to 260 mm. at the beginning of treatment. At the conclusion of treatments only 41 patients were still above 140 mm. The average fall in blood pressure was 35 mm. This lowering of blood pressure seems to be accomplished only in arthritis cases, and needs further study.

3. *Relief of Recurrent Headaches.* Six patients complained of severe headaches beginning after the onset of arthritis. In none could the etiology be determined. Of the 6, 4 have been completely relieved, 1 greatly modified, and 1 not influenced.

4. *Partial Control of Tic Douloureux.* Of 3 patients, 2 severe, 1 moderately so, the latter and 1 severe case have been free of pain for two years, and the other severe one has been kept at tolerable pain levels.

5. *Improvement of Emotional Outlook.* Probably the most impressive effect of the treatments is the realization by these patients that they are not necessarily doomed to get progressively worse and become a burden to themselves and others.

### SUMMARY AND CONCLUSION

A new treatment for the chronic arthritides is described. An analysis of 466 unselected consecutive cases, showing the types, the severity, and the results from the physician's point of view, is given. An evaluation of the treatment by 277 patients is also given. Eighty-two per cent under treatment stated that their progress was satisfactory. Ninety-three per cent of the patients whose treatments had ended from six months to five years before questioning stated that their present status was satisfactory.

This treatment has many advantages over others in current use. Briefly these are:

1. It is applicable to all types of arthritis in ambulant patients.
2. There are no contraindications and no unfavorable side-effects.
3. There is no interference with the course or the treatment of concomitant diseases.
4. The improvement is sustained, and relapses are infrequent.
5. Emergency surgery is not interfered with. Injections have been given on the day before surgery and throughout convalescence, also throughout pregnancy and lactation.
6. The medication is easy to administer and need be given only once a week.
7. The treatments are relatively inexpensive.

The general conclusion is that the treatment of arthritis with injections of cobra venom combined with formic and silicic acids offers greater possibilities for improvement and clinical arrest than other treatments currently used.

## REFERENCES

1. Albee, F. H., and Maier, E.: Preliminary report on use of venom in arthritis, M. Rec. *156:* 217 (April) 1943.
2. Steinbrocker, O., McEachern, G. C., La Motta, E. P., and Brooks, F.: Experience with cobra venom in arthralgias and related conditions, J. A. M. A. *114:* 318 (Jan. 27) 1940.

# EVALUATION OF THE BRYSON TREATMENT OF ARTHRITIS*

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After examining a group of unselected patients, who had been treated for chronic arthritis by Dr. Kenneth Bryson, of Cleveland, it seemed desirable either to confirm or to refute his claims for the effectiveness of a mixture of cobra venom, silicic and formic acids in the treatment of arthritic cases. Accordingly, a clinic was set up in the Out-Patient Department of The Maryland General Hospital for this purpose. At first only patients referred by their family physicians were accepted for study. However, as patients benefited from the treatment some became so enthusiastic that they referred other arthritics to the clinic.

This study was started with real skepticism. In an attempt to be thoroughly objective, the components of Bryson's mixture were separately prepared in sterile ampules and designated by letters, so that the person giving the injections, did not know what particular substance was being given. Solution *A* contained formic acid; solution *B* was made up of cobra venom, formic and silicic acids;† solution *C* was a mixture of formic and silicic acids.

At the time of this report the study has been in progress for 15 months, during which period 88 patients have been treated. The severity of the disease varied considerably, but in every instance was grave enough to cause the patient to seek relief and to continue therapy. The types of arthritis were simply classified as rheumatoid or hypertrophic, the latter predominating. The age of the patients varied from 34 to 85 years. Previous forms of therapy which had been tried by these patients were, salicylates, gold, cortisone, hydrocortone, ACTH and butazolidine. The response to these agents had varied, but on the whole was unsatisfactory. Before admitting any patient to the clinic for this investigation, all other medication for arthritis was discontinued, and it was required that the patients refrain from taking any adjuvant therapy while under observation.

The form of treatment employed in this study was exactly that described by Bryson,[1] namely, subcutaneous injections of increasing quantities of material at weekly intervals. It seems that the effectiveness of the medication is enhanced when the solutions are put close to the deeper layers of the dermis. In nearly every instance 1 cc. of 2% novocaine was added to the medication before administration. The initial treatment consisted of 0.5 cc. of novocaine plus 0.5 cc. of the material being tested. The weekly injections were then increased by increments of 0.5 cc. until 3 cc. of the medication was given. This quantity was the dose for subsequent treatments. When the larger doses were given it was customary to deposit the material at more than one site. On the average the maximum relief of symptoms was reached in 16 weeks, but weekly injections were continued for another month. Thereafter the frequency of treat-

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† Nyloxin. Hynson, Westcott & Dunning, Inc., Baltimore, MD.

ments was changed to once in every two weeks for four times and if the patients were improving the interval between injections was lengthened to three weeks for four treatments. In some patients the treatments were then discontinued whereas others were maintained on monthly injections. There have been 9 patients who have stopped treatments for at least five months and have maintained the improvement derived from therapy.

## Toxicity and Reactions

In none of the 88 patients studied at The Maryland General Hospital was any evidence of toxicity detected. This, however, is not surprising when it is recalled that Bryson treated almost 500 individuals with the cobra venom mixture without a single instance of severe systemic reaction. Furthermore, the quantity of cobra venom given to the arthritic patient is far below the quantities previously given to patients suffering from advanced neoplastic diseases.[2,3] In a few of the patients in this study, blood samples were taken to detect the presence of specific antitoxin but none was found. Allergic reactions were noted in 2 of the 88 patients. The first was in the form of flushing and moderately severe itching which was quickly controlled with an antihistaminic on the day of injection. In this case, the treatment with Bryson's solution was continued. The second reaction occurred in a patient with psoriasis. This condition became greatly aggravated after several injections of formic acid and was not relieved by an antihistaminic, consequently, the injections were stopped. Swelling and redness at the site of injection were noted in several patients. However, the extent of their local reactions diminished with each treatment and disappeared by the time the full therapeutic dose had been reached.

## Response

The average response to therapy began at about the sixth week of treatment. Frequently this was so slight that at first the benefit was questionable. Therefore, any patient receiving less than six treatments is not included in this report. Only in the case of the patients treated with Solution *A*—i.e., formic acid alone—did the patients feel that the therapy was of no benefit. It soon became evident that Solution *B* (cobra venom, formic and silicic acids) was far more effective than the control solutions.

The results of the study are as follows:

## Statistics of Cases Studied

Patients treated—88
Cases too early to report—16
Cases reported—72
*Solution "A"* (Formic Acid)
Patients treated—10
Patients responding—1
Patients not responding—9
One of these patients had psoriasis, which flared up with each injection, so that

treatment had to be discontinued. Another patient stopped therapy after two treatments. The remaining patients were switched to Solution "B" after twelve weeks. Five have shown definite improvement and 3 have only had three treatments with this solution and are therefore not to be evaluated.

*Solution "B"* (Formic Acid, Silicic Acid and Cobra Venom)

Patients treated—66

Of these, 4 patients stopped treatment too soon (3 treatments or less), and 1 case was a patient with a back injury whose symptoms were not known to be due entirely to arthritis. These, therefore, were dropped from the cases reported.

Cases reported—61

Patients responding—52

Patients not responding—9

Patients responding in percentage—87 per cent

*Solution "C"* (Formic and Silicic Acids)

Patients treated—10

Patients responding—8

Patients not responding—2

Of the 8 patients responding, the improvement was less than with Solution "B". At the end of 16 weeks, when no further improvement was noted, these patients were then given Solution "B", following which, improvement continued. Of the two patients not responding to Solution "C", 1 of them showed marked improvement when changed to Solution "B".

By the term "responding" we mean the subsidence or cessation of the cardinal manifestations of arthritis. These may be listed as follows:

1. Relief of pain: This effect is not limited to the particular joints involved in the arthritic process but also applies to referred pain such as occipital headache, or pain in the arms from cervical arthritis.

2. Subsidence of swelling was often the first sign of improvement.

3. Lessening of stiffness usually paralleled the diminution in pain.

4. Increased mobility; the degree of increased motion was very remarkable in some patients.

5. Improvement in the patients' sense of well being was frequently commented upon by the patients, but was thought to occur only after relief from acute symptoms had been secured.

Accurate and objective evaluation of improvement in cases of arthritis is difficult, but the judgment of a large number of chronic sufferers is something that cannot be ignored. It is a fact that more than 80 per cent of the patients in this series treated with Bryson's solution were certain that they had been greatly helped. Nine of the 61 have remained in a satisfactory state for at least five months after the cessation of therapy. Several others who had stopped injections had to resume therapy, but it is important to point out that in every instance they did so on their own request.

## Summary

The observations at the arthritic clinic of The Maryland General Hospital confirm the claims made by Bryson for the effectiveness of a mixture of cobra

venom, silicic and formic acids in the treatment of chronic arthritis. Although only 61 patients have received the full course of injections, the results parallel those reported by Bryson. No serious side reactions were observed in the patients studied. The materials used are inexpensive and require administration only once a week. It is our conclusion that the treatment advocated by Bryson is not a "cure" for arthritis, but does give substantial relief of symptoms to more than 80 per cent of the patients.

## REFERENCES

1. Bryson, K.: Treatment of chronic arthritis with a combination of cobra venom, formic acid and silicic acid, Am. Surgeon (In press).
2. Hills, R. G., and Firor, W. M.: Use of more potent cobra venom for intractable pain, Am. Surgeon *18:* 875 (Sept.) 1952.
3. Steinbrocker, O., McEachern, G., LaMotta, E. P., and Brooks, F.: Experience with cobra venom in the arthralgias and related conditions, Jour. A. M. A. *114:* 318-322 (Jan. 27) 1940.

# ESOPHAGITIS: A REVIEW

## WITH ESPECIAL REFERENCE TO EFFECTS OF SUBTOTAL GASTRECTOMY ON ESOPHAGITIS

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### INTRODUCTION

### *Case Reports*

*Case 1.* Shortly after the favorable report on gastric resection for esophagitis and stricture of acid-peptic origin by Wangensteen and Leven[182] in the May 1949 issue of Surgery, Gynecology and Obstetrics, a 23 year old woman was admitted to the surgical service at Touro Infirmary for esophagitis. She had begun with dysphagia in 1945, and since that time had been having progressive difficulty, initially with solid foods, and later with all foods and fluids. The patient was conscious of esophageal fullness, and used a Valsalva maneuver to produce emptying. There was recumbency regurgitation. At times she induced vomiting for relief of substernal pressure. The vomitus contained undigested food with a putrid but not sour odor.

A study elsewhere in 1946 led to a diagnosis of cardiospasm with secondary dilatation and she was taught to use mercury bougies, which she did until November 1948, at which time her physician did a Grondahl esophagogastrostomy (fig. 1). This procedure completely relieved her dysphagia. However, thereafter she developed dyspepsia; complained of sourness, pyrosis, regurgitation, nausea and vomiting, unless she remained on a bland diet. The initial episode of hematemesis and melena occurred four weeks after her operative procedure, and between November 1948, and May 1949, she had 12 episodes of massive hemorrhage, both with hematemesis and melena, numerous episodes of minor hemorrhage, and required 25 transfusions during that interval. She had lost 25 pounds in weight.

There was a marked hypochromic, microcytic anemia. There were 62 degrees of total acid. With barium study the cardia was much deformed (fig. 2 and 3) and the esophagogastric junction was lateral to its normal position. There was a small pouching at the esophagogastric junction which was interpreted as a penetrating ulcer. The stomach was dilated with retained food and secretions.

Passage of the esophagoscope caused active bleeding. The distal third of the esophagus was involved in a diffuse, erosive esophagitis, and there were numerous mucosal ulcerations but no deep ulceration. The esophagogastric junction was patent and the gastric contents regurgitated freely into the esophagus. During this hospitalization her response to a medical regimen was considered satisfactory.

In August 1949, she was readmitted to the hospital with a massive hemorrhage. On September 2, an anterior Polya subtotal gastric resection was done for the esophagitis.

Three weeks later there still was a reflux of barium into the esophagus from the stomach in the horizontal position, but there was no erosion in the lower esophagus. Retrograde barium reflux had diminished by Nov. 22, 1949. There were, however, some mucosal changes in the esophagus, probably as a result of esophagitis. There was diffuse spasm of the lower esophagus with some minimal mucosal changes in May 1952.

When she returned in August of that year, she stated that she frequently had been nauseated, and vomited very occasionally. There was only one time when she had had blood streaked vomitus. She was complaining of moderate dysphagia, epigastric pain, fatigue and

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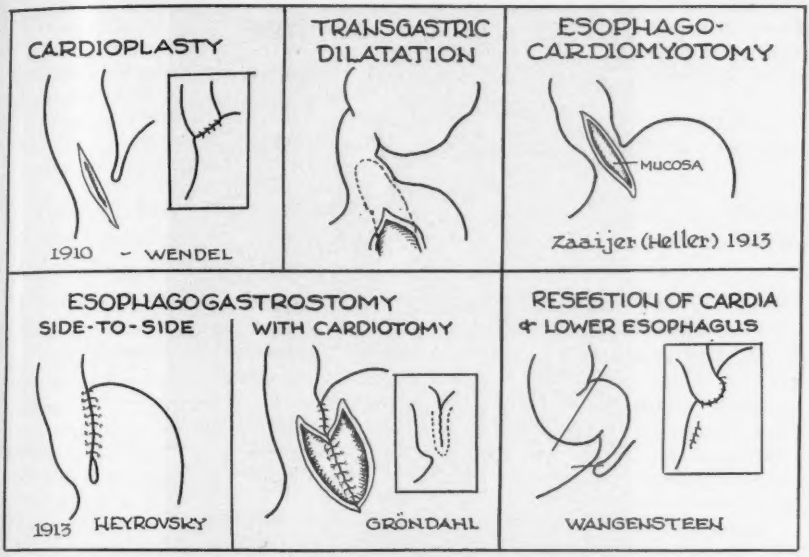


FIG. 1. Types of operation for obstruction at the lower end of the esophagus

FIG. 2. Postesophagoplasty patient with an ulcer at the cardioesophageal juncture

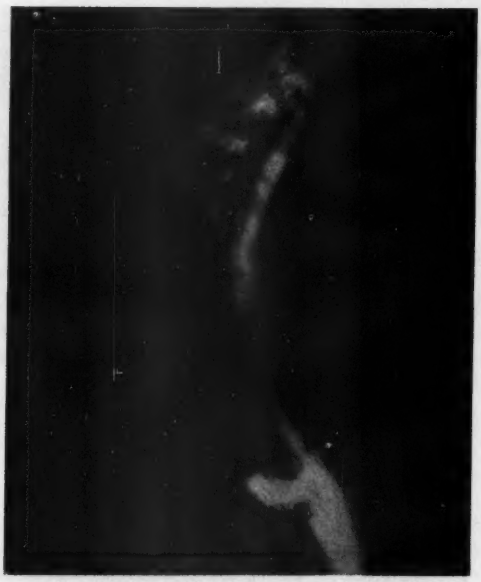

FIG. 3. Postesophagoplasty patient. This lateral view shows the deformity of the fundus of the stomach.

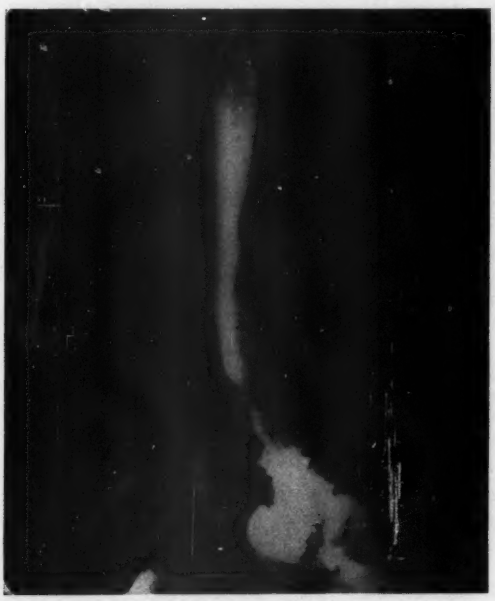

FIG. 4. Postesophagoplasty patient. The arrow in this figure shows the serrations along the lower esophagus.

weakness. The roentgenogram showed slight transitory spasm in the region of the infradiaphragmatic portion of the esophagus and in the recumbent position barium easily flowed into the lower esophagus. A small hernia had developed. There was a serrated outline of the lower half of the esophagus (fig. 4).

In August 1953, she was living in Florida and was six months pregnant. She became obstructed by a bolus of food and a gastrostomy or jejunostomy was being contemplated to tide her over her pregnancy.

*Comment:* Because of the train of events observed in this woman, the effects of subtotal gastric resection on esophagitis have been studied in my private practice and at Charity Hospital, New Orleans. Three patients upon whom I did subtotal gastric resections for duodenal ulcer, and who had definite esophagitis as an incidental finding have had no further complaints referable to this condition.

That subtotal gastric resection may make matters worse is reflected in the course of the following patients.

*Case 2.* A 37 year old woman was admitted to Charity Hospital on Sept. 5, 1950 with a four year history of intractable epigastric pain. She had a penetrating duodenal ulcer with crater formation and 50 per cent gastric retention. During her hospitalization she began to bleed, and on September 11 an emergency retrocolic Hofmeister gastrectomy was done. Obstruction of the efferent stoma developed in June 1951. There was free hydrochloric acid in the gastric remnant. No esophagitis was noted on the roentgenogram at this time. On July 3, 1951 she was explored and a gastrojejunal ulcer was found obstructing the distal stoma. A vagotomy and further resection were done and the Finney-von Haberer type of gastroduodenostomy was used to restore continuity of the alimentary tract. Postoperatively she vomited frequently and this persisted for three months. When the next roentgenogram was taken she had marked spasm at the cardia. By August 1952 the esophagus was dilated and filled with retained food particles.

The esophagus was tapered to a point and there was a delay of barium for a period of three to four minutes at the cardioesophageal junction. There was no evidence of gastric mucosa in the chest portion of the esophagus. The stomach filled and emptied satisfactorily. She was receiving esophageal dilatations with some improvement.

*Case 3.* A history of abdominal distress and epigastric pain which had been getting worse during a seven weeks period of repeated episodes prompted a 50 year old man to seek admission to Charity Hospital on Oct. 14, 1951. He had been hiccoughing for one month. When the symptomatology from a constant deformity of the duodenal bulb with pseudo-diverticulosis failed to clear on a medical regimen an antecolic Polya subtotal gastric resection was done. Four days after operation the wound separated and secondary closure was necessary. For a period of two weeks thereafter, there was vomiting from partial obstruction of the efferent stoma.

Three months later he was admitted to the hospital unable to swallow foods. There was a constant narrowing of the lower third of the esophagus. The gastrectomy stoma was functioning well. On esophagoscopic visualization severe inflammatory changes were seen and the pathologic report of a biopsy specimen was subacute and chronic esophagitis. In the next four months the stenosis progressed and frequent dilatations of the esophagus were necessary. A year later he died of a subdural hematoma.

*Comment:* So far as the records would indicate, the 2 Charity Hospital patients who developed esophagitis did so after subtotal gastric resection. The onset conceivably may have been prior to the time of operation, but there was no gross evidence of esophagitis until the patients developed complications after subtotal gastrectomy. The obstruction of the efferent stoma may be presumed to have enhanced gastrojejunal regurgitation into the lower esophagus. Later, however,

there were normally functioning gastrojejunal outlets; nonetheless, the cicatricial stenosis progressed.

These case studies dealing with esophagitis are revealing of many of the problems that concern the various specialty groups who are interested in the lower end of the esophagus and upper end of the stomach.

## ANATOMY AND PHYSIOLOGY OF THE CARDIA

The esophagus passes between the crura of the diaphragm. There are two definite bands of the right crus which encircle the esophagus and insert into the central tendon of the diaphragm (fig. 5). There is a small slip of muscle from the left crus which inserts into the central tendon around the diaphragmatic opening of the vena cava. The crura are innervated chiefly from the phrenic nerve of the same side, whether the muscles originate from the crus of the same side or the opposite[70] side. They may receive a nerve supply from the first lumbar nerve.[116] The hiatus is open during swallowing and expiration[193]. It is closed during inspiration due to the contraction of the crura and this important mechanism assists greatly in assuring continence of the cardia.

The peritoneum is reflected from the stomach onto the esophagus, and on the superior aspect of the diaphragm the mediastinal pleura is bound to the esophagus. The phreno-esophageal membrane or ligament (fig. 6) made up chiefly of fat and loose areolar tissue, gives a flexible connection between the diaphragm, esophagus and cardia. The designation of this tissue as a ligament gives a false impression of its strength.

There is an oblique entry of the esophagus into the stomach (fig. 7). This obliquity may be due in part to the tethering effect of the short left gastric artery

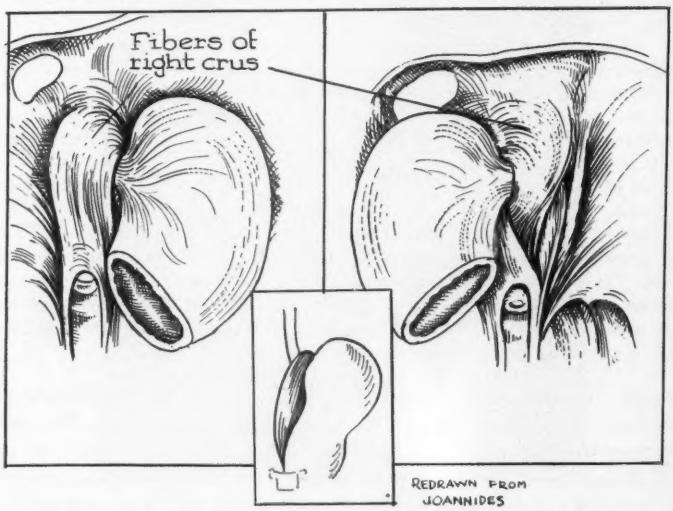


FIG. 5. The anatomy of the right crus of the diaphragm is shown

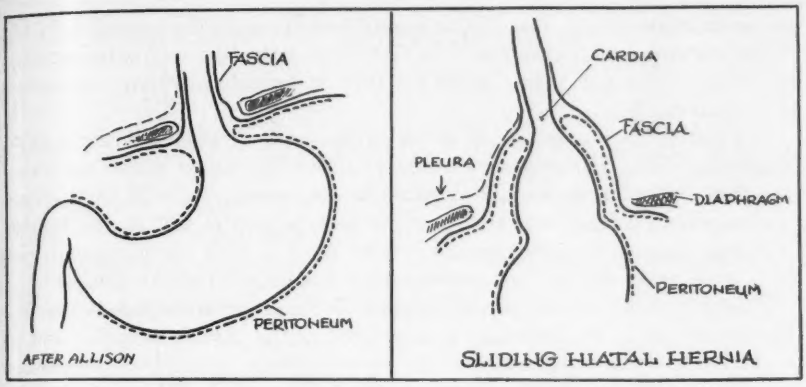


FIG. 6. The anatomy at the cardioesophageal junction

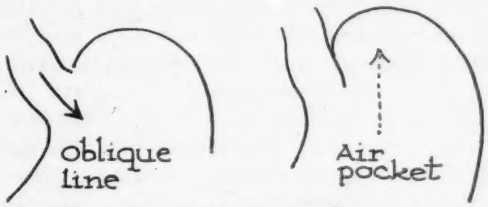


FIG. 7. Shows the oblique entrance of the esophagus into the stomach. Air in the stomach to the left of the esophagus may have a flap-valve effect.

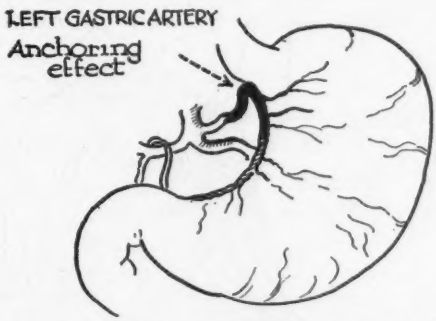


FIG. 8. Short left gastric artery. The length of this artery may play a role in the production of hernia.

(fig. 8), and the length of this artery may play a role in the production of hernia. The pressure effect of an air bubble in the stomach to the left side of the esophagus may have a flap-valve effect[103] (fig. 7).

The intrinsic musculature of the cardioesophageal area is continuous. The longitudinal muscle of the esophagus is continued down onto the stomach as a well defined layer. The circular muscle of the esophagus occasionally may be dissected

out on the stomach side. The oblique musculature is practically impossible to find on the stomach side. A collection of muscle fibers within the wall of the stomach may form a muscular collar,[11] which has been explained as causing a sphincter-like action (fig. 9).

The mucosa at the lower end of the esophagus is arranged in very narrow, longitudinal lines. Immediately past the epithelial line which marks the transition from squamous epithelium to glandular epithelium, the folds are arranged in a transverse manner, and the rugae are very large (fig. 10). In the isolated cadaveric gastroesophageal segment, water injected from the esophageal side flows more freely through the specimen than that injected at the pylorus.

There is a normal hourglass arrangement of the terminal esophagus (fig. 11.) The upper half of the hourglass is designated as the phrenic ampulla, and its length is 3 to 5 cm.; the insertion of the ascending sheet of the phreno-esophageal membrane marks its upper[124] boundary. At the lower end of the upper half, or where the constriction is in the middle of the hourglass, is the inferior esophageal sphincter. The middle constriction is also near the level of the diaphragm where the crura encircle the esophagus. The lower half of the hourglass is the gastro-esophageal vestibule. The upper end of the vestibule is formed by the inferior esophageal sphincter near the diaphragmatic opening and below by the constrictor cardia, which is at the epithelial line. The constrictor is made up of smooth muscle of the circular and spiral musculature. The phrenic ampulla is supradiaphragmatic and the gastroesophageal vestibule is below the diaphragm.

The studies of Lerche[101] indicate that the bolus of food requires four to five seconds to get to the phrenic ampulla. The bolus remains for about one-half second in the phrenic ampulla. This presumably is due to the inferior esophageal

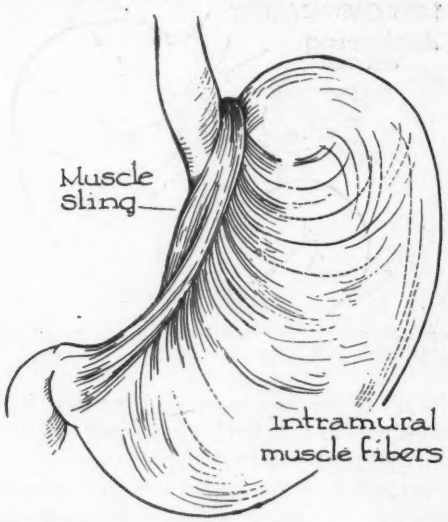


FIG. 9. Muscular fibers within the wall of the stomach may have a sphincter-like action

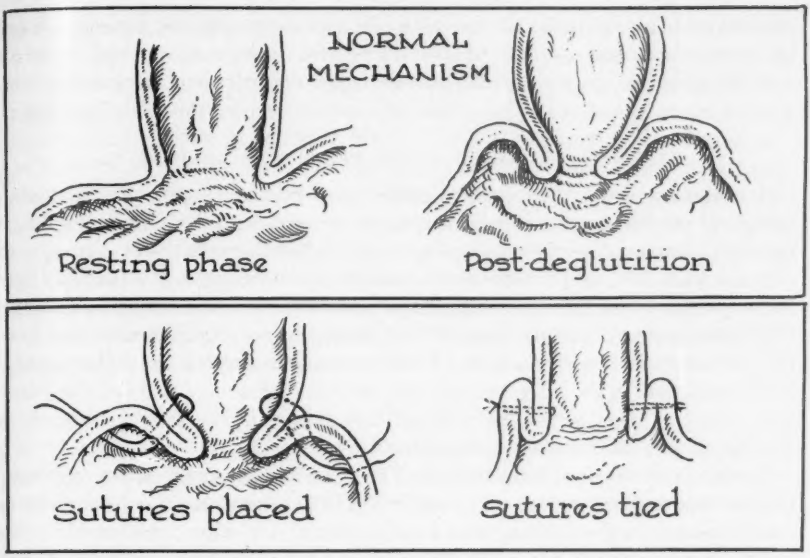


FIG. 10. Transverse rugal folds below the line of transection between the esophagus and stomach.

sphincter, and it may delay even longer if there is an overactive sphincter.[90] The upward pull on the esophagus in the act of deglutition causes the gastroesophageal vestibule to assume the form of a funnel, and contraction occurs. The constrictor cardia finally relaxes, and the mucosa around the cardiac opening intussuscepts

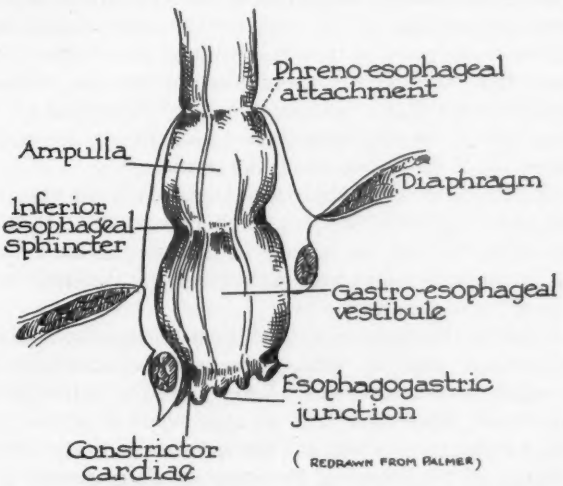


FIG. 11. Normal hourglass anatomy of the terminal esophagus

into the body of the stomach in the form of a teat-like projection, partially closing the cardioesophageal opening (fig. 10). The various components of the combined internal sphincter, that is the inferior esophageal constrictor above and the constrictor cardia below, may play a role by contracting after the bolus has passed.

### EXPERIMENTAL EVIDENCE CONCERNED WITH ESOPHAGITIS

A pathologic state very closely similar to peptic esophagitis in the human being was produced in rats by occluding the pylorus with a circumferential ligature and thereby permitting acid gastric juice to flow into the lower esophagus.[154] A dog with an occluded pylorus given histamine in beeswax over a period of time develops many erosions, ulcerations, and perforation of the esophagus, while the stomach remains relatively normal.[181] In various other experiments it has been shown that gastric juice produces a much more significant change in the esophageal mucosa than in the intestinal mucosa. So does diversion of bile and pancreatic juice into the lower esophagus, although the changes do not take place nearly as rapidly as with gastric juice bathing the lower esophagus.[38]

Gastric juice obtained from the pouch of a dog and dripped into the esophagus of a cat leads to perforation very rapidly.[177] If the same quantity of hydrochloric acid is placed on the esophagus of a cat—without the other constituents of the gastric juice—a much greater length of time will be required to effect a change. Since hydrochloric acid per se has a meager effect on the esophagus, it would seem that the changes depend upon the peptic activity of the gastric juice in an acid medium.[181]

Transplantation of a small strip of gastric mucosa into the upper esophagus produced an esophagitis especially severe in the lower third, and subtotal gastrectomy failed to relieve the esophagitis in dogs.[6] There was no difference noted in the clinical course of corrosive esophageal strictures in groups of gastrectomized and nongastrectomized dogs.[141] This tendency to cicatricial stenosis in corrosive esophagitis is much decreased by the administration of cortisone.[144] If suppurative complications which may follow cortisone administration are controlled by penicillin, perforation is not nearly as likely to occur.[145] Segmental stricture isolated from the remainder of the esophagus forms a cyst and the size of the cyst is self limited irrespective of the treatment of that segment.[44]

In dogs with the chest closed electrical stimulation of the vagus nerves shortened the esophagus and pulled it up to the diaphragm. With the chest open the stomach was pulled through the hiatus.[136] Stimulation of the various upper abdominal organs resulted in shortening of the esophagus. Vagal section or atropine abolished the reflex shortening.[46]

Increased intracranial pressure, produced experimentally, had no effect upon the gastroesophageal segment, unless there were accompanying retching and vomiting in coma or under anesthesia.[59] Neither does the hydrostatic phenomenon seem to play a part, since more than an atmosphere of pressure is required to perforate the esophagus of a dog and 80 millimeters of mercury pressure will cause perforation of the fundus of the stomach. Four pounds of pressure per square inch is necessary to produce rupture of the esophagus in the cadaver.[183]

Rupture of the esophagus experimentally occurs as a vertical tear in the lower third, and the left side is more commonly affected.[83, 13] Preceding attacks of local esophagitis may in some way contribute to the weakness of the esophagus in the lower third.[128, 115, 94]

In the dog, the Heller procedure done with a 4 inch longitudinal incision at the lower end of the esophagus and the Grondahl operation (fig. 1) cause moderate to severe esophagitis. The Heller operation, with a 2 inch incision, causes much less esophagitis. Esophageal complications are increased by vagotomy in these same animals. Reduction in gastric volume and acidity from the vagotomy probably is made up for by the increased esophageal reflux due to gastric atony, functional pyloric obstruction, and esophageal dysfunction. These surgically produced lesions in the lower esophagus are made worse by histamine in both the vagotomized and nonvagotomized animals.[66]

### ESOPHAGITIS WITH ESPECIAL REFERENCE TO REGURGITATION

Knowledge concerning peptic ulcer of the esophagus extends over a long period of time. Peptic esophagitis was described by Winkelstein as a new clinical entity in 1935.[189] Since incompetence of the cardiac sphincter is the major factor in the production of esophagitis, the terms reflux and regurgitant esophagitis more properly have been applied to this condition. In the event that the terminology of peptic esophagitis and peptic ulcer of the esophagus is used, these two terms should apply to stages in the same disease, rather than delineate them as separate entities as Barrett has done.

The most common disease of the esophagus is esophagitis.[175] Jackson[81] noted peptic ulcer of the esophagus in 88 of 4,000 autopsies. Esophagitis occurred in 7.02 per cent of 3,032 autopsies and surgery had been done in 74.6 per cent of those patients with esophagitis.[30] In 34.7 per cent of this group there had been vomiting or gastric intubation, or both, prior to death. Esophagitis most often is seen in people of middle age.[160]

Peptic esophagitis and stricture formation have been ascribed to a number of causes: the interbrain,[40, 60, 93] hiatus hernia,[3, 17, 35, 75] frequent vomiting,[82] especially during pregnancy,[174] and duodenal ulcer.[18, 20]

The combination of severe peptic duodenal ulcer and peptic esophagitis is shown in the case report of Scott[153], in which barium very readily came from the mouth of the patient, after barium enema, due to gastrojejunocolic fistula and esophageal reflux.

Rarer causes of esophagitis are pemphigus[19], arterio-mesenteric obstruction of the duodenum[138], aureomycin therapy[39], and scleroderma[36]. Thrombophlebitis within or without the esophagus may lead to esophagitis[114]. Dilatation of the cardioesophageal opening from varices forms another local factor predisposing to ulceration[158]. Patients with esophagitis may and usually have a normal acidity[21].

Esophagitis and esophageal obstruction in infants may be due to stress[69], aberrant gastric mucosa in the esophagus[139], fungus infection[33], trauma from stomach tube[56], corrosives[74], cardiochalasia, and other causes.

There has been considerable controversy over autopsy findings in esophagitis as to whether the changes take place during life; during the agonal phase, or after death. If, in the course of vomiting, there is any amount of residual gastric juice in the lower esophagus, digestion takes place.[133] The reason why digestion of the lower esophagus does not occur normally is because the gastric juice with a pH of 2[190] cannot get to the esophagus. Mucus, which acts to divert mechanical irritants and prevent chemical penetration,[29] is present in less amount in the esophagus than in the stomach.

Peptic esophagitis usually is limited to the lower half of the tube[159] and more particularly, immediately above the cardiac orifice.[159] Grossly, the types may be divided into phlegmonous and pseudomembranous; irregular and superficial coalescing ulcerations with hemorrhage or with bile staining; linear or longitudinal ulcerations with hemorrhage or black eschar; simple erosions, petechia-like lesions with congestion; and perforations and ulcerations of varying sizes and shapes.[14] At an early stage the mucosa is a fiery red, and has been likened to the skin around a gastric fistula.[10] Minute ulcerations develop, and the bordering mucosa is inflamed. As the disease proceeds, the superficial ulcerations coalesce, and later there is penetration of the deeper layers of the esophagus. The ulcers may become very large. One ulcer 20 cm. in diameter has been reported.[146] The ulcer may penetrate and fix itself, or it may perforate into the mediastinum,[132] or into the pleural cavity.[32] Stricture formation ensues and the constriction may be a narrow short segment or involve most of the esophagus.

Franklin[63] has recently described a nonspecific regional granulomatous esophagitis with a histologic picture similar to regional enteritis.

The diagnosis by roentgenogram of various constricting lesions in the lower third of the the esophagus is suggestive of esophagitis when there is a wooliness of the mucosa, and expansibility of the stenosis.[84] Any diagnosis made tends to be diagnosis by exclusion.[131] There is a fairly elongated, funnel shaped constriction in the distal part of the esophagus, and the upper limit of that constriction is, in addition,relatively distinct, giving the impression of a horizontal upper limit. In all cases there is a finely notched irregular relief from membranous or pseudomembranous coating on the inflamed mucous membrane.[184] There may be intermittent diffuse spasm of the lower half or lower third of the esophagus.[127]

There are a number of constricting lesions in the lower third of the esophagus which are impossible to interpret by roentgenogram and esophagogastric biopsies, and these will require resection of the segment in order to establish a diagnosis.[21] Once stenosis of the esophagus has occurred from peptic esophagitis, all forms of treatment are to a lesser or greater degree unsatisfactory. The esophagus is shortened and restoration of the normal hiatal mechanism is impossible. Dilatations may help. Various surgical methods have been proposed. A conservative approach is that of Thorek[170] who has recommended a temporary external esophageal fistula over a T tube. Another conservative surgical approach is to do a local excision of an isolated narrow stricture and anastomose the upper end of the esophagus to the stomach or to the esophagus.[91]

Local excision was employed in a 60 year old white woman, who was seen in

FIG. 12. Stricture of the lower end of esophagus

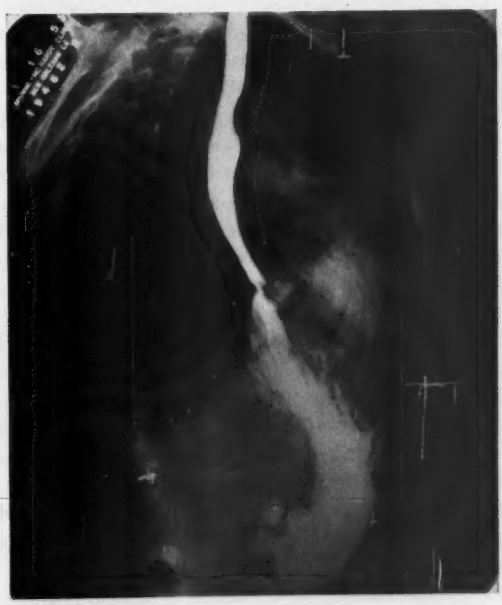

FIG. 13. Early postoperative film after local excision

July 1953. She gave the history of repeated esophageal dilatations over a four year period, but despite dilatations she had lost 40 pounds. Roentgenogram (fig. 12) and esophagoscopy proved a narrow stricture of the esophagogastric junction with a small projection of gastric mucosa above the diaphragm. This short stricture, without any evidence of acute esophagitis, was resected on July 8, 1953 as recommended by Sweet[165] for this type of esophageal obstruction. An esophagogastric anastomosis was done without an extensive resection. It was not feasible to bring the anastomosis below the diaphragm. According to her last roentgenogram (fig. 13) there is satisfactory function of the anastomosis.

The severe forms of esophagitis require a more radical resection, and, while earlier reports advocated a wide resection of the esophagus,[62, 171, 188] the emphasis now is on removal of most of the stomach to cut down on acid peptic activity,[172, 191] and either do an esophagogastrostomy or, as Allison[3a] and others suggest,[9] partial esophagogastrectomy and esophagoenterostomy, employing the Roux Y type of anastomosis.

## HIATUS HERNIA

Hiatus hernia is the most common type of diaphragmatic hernia[142] in adults, and second in frequency to the hernia of Bochdalek[148, 98] in children.

One and one-half per cent of normal, asymptomatic people have hiatus[34] hernias. Obesity[50] and old age[8] are predisposing factors. Pregnancy often is complicated by diaphragmatic[102, 187] hernia and 18.1 per cent of multipara in the third trimester of pregnancy had hiatus hernias[140a]. There may be a coexisting gastric ulcer and carcinoma[57, 58]

The symptoms of diaphragmatic hernia are related to the associated esophagitis. Pain in the substernal retrosternal area which radiates to the neck, and occasionally to the back, often is made worse by bending forward or lying down. Pain during a meal is said to occur only when an ulcer is present.[47] Significant ulceration occurs in 60 per cent of hernias of the short esophagus[121] type. Dyspepsia is another common symptom.[163] Dysphagia and vomiting[79] occur. Many patients develop anemia from bleeding of the mucosal ulcerations.[22] Hiatus hernias may masquerade as coronary disease or various other forms of upper abdominal[166] pathology. Many patients are branded as psychoneurotics.[15]

The diagnosis of hiatus hernia is made by roentgenogram, and it is being made more frequently.[86] The phrenic ampulla must be distinguished from hiatal hernia.[112] The gastric mucosa must be identified above the diaphragm in a hiatal hernia, and hypertrophy of the gastric mucosa helps make the diagnosis.[16] A projection to the left side seen immediately above the diaphragm also should be considered a hernia, especially if the greater curvature of the stomach just above the cardia presents a concave border when examined erect.[85] The left anterior oblique recumbent position also is recommended as being of value in bringing out these findings.[69a] Esophagoscopy is important in the differential diagnosis of hernia and as a guide in the treatment.[110]

Hiatus hernia can be likened to an inguinal hernia. There is a hole in the abdominal wall; there is a normal structure passing through the aperture, and

there is increased intra-abdominal pressure. There are three classical[2] types, the esophagogastric (sliding) type, the paraesophageal (rolling) type, and thoracic stomach or short esophagus. There is essentially little difference in the gastroesophageal and thoracic stomach type except that in the former there is curling of the esophagus and no loss of length, and in the latter the esophagus is short. Usually the shortness is apparent rather than real.[150] Probably most shortening of the esophagus is due to esophagitis, and fibrosis, especially contraction of the longitudinal muscle fibers in the esophageal[157] wall, and not due to congenital shortening. In the esophagogastric hernia, the stomach projects squarely through the hiatus into the chest, and there is relaxation of its various attachments. The gastroesophageal juncture is intrathoracic and there is no sac. The paraesophageal type is a herniation of the stomach along the side of the hiatus and the normal cardioesophageal line is maintained intact. In the paraesophageal type there is a sac and the circumferential ligamentous attachments of the cardioesophageal juncture are intact. Once the paraesophageal hernia begins to enlarge, the attachments around the diaphragm often are not sufficiently strong to support it in that position, and there is then a combination of the two types of hernia. When this occurs in the paraesophageal type, the sphincter mechanism of the cardia is lost; reflux esophagitis occurs in this combination type of hernia. It may be difficult on the roentgenogram to see the upward extension of the cardioesophageal line because of the size of the paraesophageal bulge which may obscure the area.

The cardia must be abdominal, and the nervous co-ordination between the internal and external muscle mechanisms operating at this level must be intact. If there is displacement upward of the cardioesophageal juncture—and this is what obtains with hiatal hernia—the crura are ineffective in obstructing the opening during inspiration and in supporting the intramural movements. As a result, there is serious interference with the delicate closing mechanism around the cardia.[176]

Surgical treatment of hiatus hernia is now undergoing the technical evolution that inguinal hernioplasty went through toward the close of the last century.[149] The problem of esophagitis is an added factor. There are undoubtedly a number of cases in which peptic gastroduodenal ulceration with pyloric obstruction antedate the esophagitis and the hiatal hernia. In such a situation the hiatus hernia must be secondary. If this be true, repair of the hernia will accomplish very little.

The purpose of the surgical correction of hiatal hernia is to restore, insofar as is possible, the normal relationship at the cardioesophageal aperture. To do this various methods are in use. The method of Allison,[3a] by way of the thoracic approach, or one of its modifications frequently is employed.[49, 48, 88, 99a] The technic of Allison is to tack the fibro-fatty ring around the esophagogastric juncture to the undersurface of the diaphragm, and to bring the crura together behind the esophagus. Sweet[162] plicates or excises the sac. He also brings the crura together behind the esophagus. Harrington[71] considers the repair can be accomplished from the abdominal route, and in addition crushes the phrenic nerve. Other technics have been described; as moving the cardioesophageal hiatus;[116, 179] enlarging

TABLE I

TRANSTHORACIC HIATAL HERNIA REPAIR

CHARITY HOSPITAL 1937–1953

| | FOLLOW-UP | ESOPHAGITIS | | RECURRENCE |
|---|---|---|---|---|
| | | SYMPTOMS | X-RAY | |
| ALLISON REPAIR | 7 months | Mild | Negative | No |
| SHIFT HIATUS | 1 year | Severe | Positive | Yes |
| | 3 months | " | " | " |
| | 5 months | " | " | No |
| | 5 months | Mild | Negative | " |
| | 1 year | None | " | " |
| | Died postoperatively | | | |
| TANTALUM | 1 year | Mild | Positive | Yes |

the hiatus and suturing the margin of the stomach to the diaphragmatic defect,[85] and phrenico-exeresis.[96] These measures lead to esophagitis.

Recently Hays[73] has described a method of invaginating the esophagus into the jejunum (fig. 10) in doing an esophagojejunal anastomosis, in order to prevent regurgitation into the esophagus. This suture technic closely resembles the normal deglutitory mechanism at the cardia during the so-called intussusception phase after expulsion of the bolus from the phrenic ampulla (fig. 10). This added step to the Allison repair would increase the danger of contamination since the stomach would have to be opened to facilitate placement of sutures. It could also increase the danger of obstruction, but it may prove worthwhile to aid in the prevention of reflux. As is shown in table I, the hernia may not recur, and yet, the esophagitis may prove troublesome.

### ACHALASIA

What happens to the esophagus in achalasia? It is believed by some[89, 90] that Auerbach's plexus is wanting; sympathetic control predominates; a tight sphincter develops at the cardioesophageal juncture, and the enlargement and hypertrophy of the proximal esophagus which follow are secondary to obstruction at this point. Others[164] believe that the lower end of the esophagus becomes an atrophic segment, and normal motility does not carry across this segment, producing a functional obstruction. Another theory concerning the etiology is phrenospasm.[135] Wangensteen[178] likes the term dystonia of the esophagus to depict the derangement.

Whether or nor cardiospasm results occasionally from carcinoma is undecided.[7] Diverticula[44a], cardiochalasia in infancy[41], curling of the esophagus[54], wandering[78], or corkscrew esophagus[80, 109], functional tense esophagus[95], or tertiary contractions[84] or abnormally long projections below the diaphragm[147] probably represent types of nervous imbalance. The role of the globus pallidum in the act of deglutition has been stressed in relation to segmental spasms of the esophagus and Parkinsonism.[129]

The pathologic anatomy in achalasia is a fusiform dilatation of the esophagus from the cardioesophageal juncture to the thoracic inlet. There is normal narrowing of the esophagus at the hiatus. There is mild irregular thickening of the muscle coat at the cardioesophageal juncture and at an interval just above it,[42] probably representing enlargement of the constrictor cardia and the inferior esophageal sphincter respectively. The myenteric plexus is partially or totally absent well up the esophagus and there is replacement by capsular and interstitial supporting cells.[37]

Many patients with cardiospasm also have various types of pulmonary pathology due to regurgitation of esophageal contents into the bronchial tree.[4, 68, 99]

As a result of the advancements in surgery of the esophagus,[107, 45] many patients with achalasia have been operated upon during the last 15 years.[106, 118, 120, 160, 180] Vinson[173] and others[122, 111] believe that cardiospasm is much more satisfactorily handled by dilatations. The methods in use now are not of recent origin. (Fig. 1.)

FIG. 14. Achalasia

FIG. 15. Results of early postoperative Heller procedure for achalasia

Peptic esophagitis seems to be more common after plastic procedures for achalasia than for surgery in any of the other conditions in the lower end of the esophagus.[165] Many unfavorable late results have been reported with cardioplasty and esophagogastrostomy.[12, 141a, 151]

The Heller procedure, which is an extramucosal myotomy, and which preserves for the most part the anatomic relationship of the cardia, is advocated by many.[67, 27, 192, 5, 72, 168] The results in 2 patients follow. This 61 year old man was first seen in August 1952 with a 15 year history of dysphagia and intermittent periods of bouginage during that time. He had lost 50 pounds in weight during the last year. On barium swallow the esophagus was found much dilated and completely atonic with the lower esophagus narrowed a length of approximately 5 cm. Dilatation was not observed in the narrowed segment during fluoroscopy (fig. 14). He refused bouginage. On Aug. 18, 1952 a transabdominal Heller procedure was done and a longitudinal slit was made for a distance of about 10 cm. along the esophagus. Dysphagia completely disappeared following the Heller procedure and a month later there was a definite decrease in the degree of dilatation of the esophagus as seen on the esophagogram (fig. 15). He gained 25 pounds by May 1953, but was beginning to have mild dysphagia at that time and there was a greater delay in the complete emptying of the lower esophagus with barium ingestion (fig. 16). In view of these findings, a dilatation was done.

Another case report, that of a 39 year old woman who was admitted to Charity Hospital in November 1950 has essentially the same story. A Heller

FIG. 16. Appearance of esophagus one year after Heller procedure for achalasia

procedure was done on Dec. 1, 1950 and the operative note stated that excellent dilatation of the cardioesophageal narrowing was obtained. Seven months later, there was narrowing again at this point, and there was retention of mucus and food particles and chronic inflammatory reaction involving the lower three quarters of the esophagus. Dilatations were started on admission with excellent results.

Two experiences with the Heller procedure are not enough to condemn it. However, certain facts stand out. If the Heller procedure affords a patulous orifice there results an esophagitis. As good results, with anything less than an extensive splitting of the cardioesophageal musculature can usually, but not always, be obtained, in good hands, by dilatation from above. The Heller procedure is an excellent procedure, provided its shortcomings are appreciated. Just as 25 per cent of our patients with achalasia have difficulty after dilatations, so a surgical procedure which accomplishes the same thing can be expected to have a number of recurrences. Be that as it may, a patient well can afford to have a second Heller operation in order to escape reflux esophagitis which follows on the heels of other procedures used in the correction of achalasia (table II).

### ESOPHAGITIS FOLLOWING RESECTION FOR TUMOR

From the time of Finney's report[61] on total gastrectomy until the present, numerous reports[137, 156, 185, 186, 26, 169, 155, 152, 126, 1, 51, 134, 125, 52, 130] have given evidence of the improved surgical results in the handling of malignant and benign

TABLE II

SURGICAL MANAGEMENT OF ACHALASIA

CHARITY HOSPITAL 1937-1953

| PROCEDURE | FOLLOW-UP | RESULT |
|---|---|---|
| WENDEL | 2 years | Stricture |
| | 4 years | " |
| | 1 month | " |
| | 2 months | Inflammation |
| | 7 years | Asym |
| HELLER | 1½ years | Required dilatation |
| GRONDAHL | 1 year | Esophagitis |
| HEYROVSKY | 1 year | " |
| | 5 years | " |
| | 1 year | Severe esophagitis |
| | 4 months | Dilatation |
| | 1 year | Pulmonary symptoms |
| ESOPHAGECTOMY | No follow-up | |

tumors in this region. There still are numerous opportunities for further development and improvement,[104] notwithstanding the surgical advances, for mortality and morbidity figures still are high[55, 119] and the results of irradiation therapy, including rotation irradiation for esophageal lesions, have not been encouraging.[64, 152]

One of the disabling complications which often follows successful total gastrectomy, or resection of the esophagus for benign or malignant tumors, is reflux esophagitis[130] resulting in semi-invalidism. After total gastrectomy many ingenuous procedures[123, 73, 87, 43, 53, 76, 77, 97, 105, 108, 113, 143, 167] have been tried to create an artificial pouch, but it is doubtful if any of these will prevent esophagitis from occurring. Esophageal grafts, large bowel and various plastic materials used as substitutes for the esophagus,[23, 24, 92, 31, 117] have as a merit avoidance of reflux esophagitis by permitting the normal cardia-hiatal mechanism to remain intact, and thereby avoiding this dreaded complication (fig. 17).

An example of progressive esophagitis following esophagogastric resection and anastomosis is illustrated by the following record: a 63 year old woman first was seen in March 1951 with a large leiomyoma of the lower end of the esophagus. This tumor was resected on March 5, 1951. A barium swallow three weeks later entered the stomach without delay, and the stoma at the esophagogastric junc-

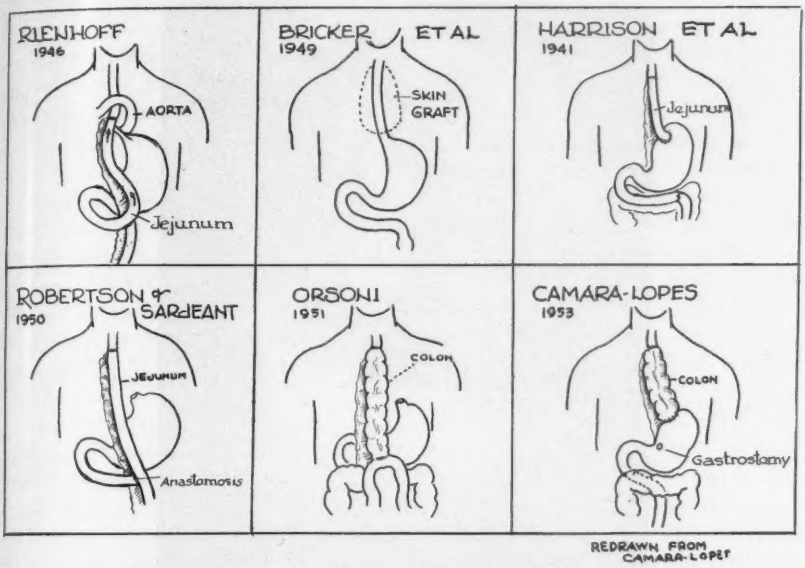


FIG. 17. Various technics used for resection of the esophagus

FIG. 18. Early postoperative appearance of esophagus after esophageal resection for tumor.

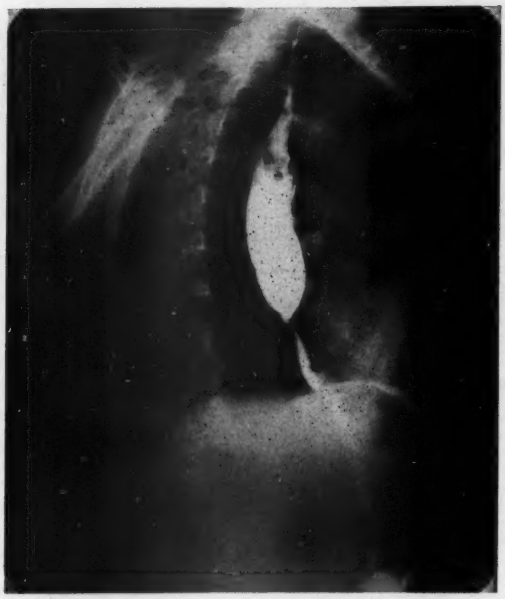

FIG. 19. Late (two and one-half years) postoperative appearance of esophagus after esophageal resection for tumor.

tion was wide, being about 3 cm. in diameter (fig. 18). A month later there was some secondary spasm, at the diaphragm, and there was fairly definite gastritis in the stomach portion. There was a mild area of erosion just above the anastomosis. There was a very wide opening at the esophagogastric junction. She was seen on Nov. 18, 1953 and at that time she had mild symptoms of esophagitis. On gastric analysis there was no free hydrochloric acid found in the stomach by histamine stimulation. Esophagogram was done and there was definite, moderate dilatation of the esophagus. There was a persistent narrowing and rather moderate spasm in the lower portion of the remaining esophagus just above the anastomosis with the stomach (fig. 19). During the delay in passage of barium a reverse peristalsis was noted. Barium regurgitated in small quantities into the lower esophagus. These findings were believed to be due to an esophagitis.

### CORROSIVE ESOPHAGITIS

Chemical burn is the most common cause of esophagitis and stricture.[25, 161] Caustic strictures usually are treated best[100, 140] with dilatations. In caustic burns of the esophagus treated by resection of the involved segment, there has been no instance of esophagitis in 13 patients in whom esophagogastrostomy was done by Burford and associates.[28] They believe that surgery for this type of esophageal obstruction may not eventuate in esophagitis and their follow-up convincing. Despite this, in view of the sequel of events in all other types of resection, it remains to be seen whether these patients remain free of esophagitis.

SUMMARY

The cardioesophageal mechanism, which provides an effective barrier to retrograde flow of gastrointestinal contents, is a very delicate and intricate one. Of the components in the makeup of this arrangement, the most important are the crura which encircle the esophagus to block it off during inspiration. The phrenoesophageal membrane, the left gastric artery, the collar of intramural muscle fibers extending around the cardia from the lesser curve of the stomach, aid in maintaining an oblique course from the esophagus to the stomach. The air bubble in the stomach may heighten the obliquity of the cardioesophageal opening producing a trap-like valve. The difference in mucosal patterns in the esophagus and stomach, combined with the expulsion force and sphincteric action of the inferior esophageal constrictor and constrictor cardia during the last phase of deglutition, may permit prolapse of gastric mucosa into the breach and hold the recently ingested contents within the stomach.

In the experimental animal pyloric obstruction leads to esophagitis. The esophageal mucosa is more sensitive than the gastrointestinal mucosa to stomach and small bowel contents, the squamous epithelium of the esophagus being especially affected by gastric juice. This sensitivity to gastric contents is due in part to its peptic activity, since hydrochloric acid alone has a meager effect upon the esophagus. Gastrectomy does not protect against esophagitis and vagotomy makes it worse. Shortening of the esophagus occurs with vagus stimulation and is abolished by vagus section.

The frequent association of reflux esophagitis and hiatus hernia is usually due to derangement of the cardia-hiatus synchronism by the hernia, with esophagitis resulting from regurgitation. Reflux in pyloric obstruction, cardia operations, vomiting, gastric intubation, and other conditions promote esophagitis which shortens the esophagus and hernia becomes manifest as a secondary phenomenon. Hernia, which is primary in origin, is best treated surgically. Unfortunately, recurrence is common after repair. It may be slow in developing and follow-up on repair must be assessed over a long period.

The best surgical treatment of achalasia is the Heller procedure. An extensive Heller operation invites regurgitation and a more timid approach invites recurrence.

Operations for benign and malignant tumors of the gastroesophageal segment, which disturb the cardia, are followed by esophagitis. Esophagitis is reported not to be a sequel of operation for corrosive stricture.

Early peptic esophagitis is best managed by correcting the cause. A narrow stricture may be excised without extensive gastrectomy. After fibrosis and shortening of the esophagus has taken place, no treatment is completely satisfactory. A wide resection of the stomach and area of stricture must be done and continuity of the alimentary tract restored, preferably by the Roux Y method.

Subtotal gastrectomy for minimal acute esophagitis, in which there is duodenal ulcer and pyloric obstruction and no other abnormality to which the esophagitis can be ascribed, may be expected to yield beneficial effects. I have 3 such patients who have shown amelioration of their symptoms and signs with this procedure. If the esophagitis is well advanced, subtotal gastric resection will not benefit the

condition. If there is obstruction at the efferent stoma of the gastrojejunal loop of the gastrectomy, esophagitis will be made worse and complete obstruction of the gullet follows.

Mechanical factors are of prime importance in the causation of esophagitis. Excepting corrosives, chemical factors assume a secondary role in etiology. Therefore, reflux or regurgitant esophagitis more accurately describes the condition than does peptic esophagitis, acid peptic esophagitis, or peptic ulcer of the esophagus.

## REFERENCES


1. Adams, R., and Hoover, W. B.: Benign tumors of esophagus; report of 3 cases, J. Thoracic Surg. *14:* 279 (Aug.) 1945.
2. Akerlund, Ake: Hernia diaphragmatica hiatus oesophagei, Acta radiol. *6:* 3, 1926.
3. Allison, P. R.: Peptic ulcer of esophagus, J. Thoracic Surg. *15:* 308 (Oct.) 1946.

3a. Allison, P. R.: Reflux esophagitis, sliding hiatal hernia, and anatomy of repair, Surg., Gynec. & Obst. *92:* 419 (April) 1951.

4. Andersen, H. A., Holman, C. B., and Olsen, A. M.: Pulmonary complications of cardiospasm, J. A. M. A. *151:* 608 (Feb. 21) 1953.
5. Anttinen, J. E.: Surgical treatment of achalasia of esophagus, Acta chir. scandinav. *103:* 442 (Fasc. 6) 1952.
6. Arroyave, R., Clatworthy, H. W., and Wangensteen, O. H.: Experimental production of esophagitis and esophageal ulcers in dogs. Forum (1950), 1951, pp. 57.
7. Asherson, N.: Cardiospasm: Intermittent: An initial manifestation of carcinoma of cardia, Brit. J. Tbc. and Dis. Chest *47:* 39 (Jan.) 1953.
8. Atkinson, A. K., and Layne, J. A.: Diaphragmatic hernia, Medical Times *81:* 89 (Feb.) 1953.
9. Barnes, W. A., and McElwee, R. S.: Surgical treatment of non-neoplastic lesions at esophago-gastric junction, Ann. Surg. *137:* 523 (April) 1953.
10. Barrett, N. R.: Chronic peptic ulcer of oesophagus and "oesophagitis," Brit. J. Surg. *38:* 175 (Oct.) 1950.
11. Barrett, N. R.: Hiatus hernia, Proc. Roy. Soc. Med. *45:* 279 (May) 1952.
12. Barrett, N. R., and Franklin, R. H.: Concerning unfavorable late results of certain operations performed in treatment of cardiospasm, Brit. J. Surg. *37:* 194 (Oct.) 1949.
13. Barrett, N. R.: Spontaneous performation of esophagus, Thorax *1:* 48 (March) 1946.
14. Bartels, E. C.: Acute ulcerative esophagitis, Arch. Path. *20:* 369 (Sept.) 1935.
15. Baum, L. F., and Baum, O. S.: Hiatus hernia, J. Med. Soc. New Jersey *50:* 91 (March) 1953.
16. Bendick, A. J.: Roentgen diagnosis in esophageal diseases, Rev. Gastroenterol. *20:* 492 (July) 1953.
17. Benedict, E. B.: Benign stricture of esophagus, Gastroenterology *6:* 328 (April) 1946.
18. Benedict, E. B., and Daland, E. M.: Benign stricture of esophagus complicating duodenal ulcer, New England J. Med. *218:* 599 (April 7) 1938.
19. Benedict, E. B., and Lever, W. F.: Stenosis of esophagus in benign mucous membrane pemphigus, Ann. Otol., Rhin., & Laryng. *61:* 1120 (Dec.) 1953.
20. Benedict, E. B., and Sweet, R. H.: Benign stricture of esophagus, Gastroenterology *11:* 618 (Nov.) 1948.
21. Benedict, E. B., Sweet, R. H., and Robbins, L. L.: Case #33501, Mass. Gen. Hosp., New England J. Med. *237:* 919 (Dec. 11) 1947.
22. Berger, S. S.: Bleeding from gastrointestinal tract, Rev. Gastroenterol. *22:* 398 (June) 1953.
23. Berman, E. F.: The plastic esophagus, J. Internat. Coll. Surgeons *18:* 695 (Nov.) 1952.
24. Berman, E. F.: A plastic prosthesis for resected esophagus, Arch. Surg. *65:* 916 (Dec.) 1952.
25. Boros, E.: Relationship of esophagus to heartburn, Rev. Gastroenterol. *20:* 132 (Feb.) 1953.
26. Brown, C. H., and Kane, C. F.: Carcinoma of stomach, Gastroenterol. *20:* 64 (Sept.) 1952.
27. Buckles, M. G.: Present status of esophageal surgery, J. Internat. Coll. Surgeons *18:* 718 (Nov.) 1952.
28. Burford, T. H., Webb, W. R., and Ackerman, L.: Caustic burns of esophagus and their surgical management; a clinico-experimental correlation, Ann. Surg. *138:* 453 (Sept.) 1953.

29. Butt, H. R., and Vinson, P. P.: Esophagitis; anatomy and physiology and review of literature, Arch. Otolaryng. *23:* 391 (April) 1936.
30. Butt, H. R., and Vinson, P. P.: Esophagitis; a pathologic and clinical study, Arch. Otolaryng. *23:* 550 (May) 1936.
31. Camara-Lopes, L. H.: Intrathoracic use of large bowel after subtotal esophagectomy for cancer, J. Thoracic Surg. *25:* 205 (Feb.) 1953.
32. Casper, N.: Rupture of esophageal ulcer, Kentucky M. J. *45:* 201 (June) 1947.
33. Castellanos, A., Codinach, A., and Pereiras, R.: Esofagitis en un nino, Arch. de med. inf. *14:* 175 (July–Sept.) 1945.
34. Cernock, W. F.: Incidence of asymptomatic hiatus hernia, Am. J. Digest Dis. *20:* 123 (May) 1953.
35. Chamberlin, D. T.: Peptic ulcer of esophagus, Am. J. Digest. Dis. *5:* 725 (Jan.) 1939.
36. Clark, D. E., and Adams, W. E.: Transthoracic esophagogastrostomy for benign strictures of lower esophagus, Ann. Surg. *122:* 942 (Dec.) 1945.
37. Cross, F. S.: Pathologic changes in megaesophagus, Surgery *31:* 647 (May) 1952.
38. Cross, F. S., and Wangensteen, O. H.: Role of bile and pancreatic juice in production of esophageal erosions and anemia, Proc. Soc. Exper. Biol. & Med. *77:* 862 (Aug.) 1951.
39. Curtis, V. J., Giliberti, J. J., and Schraft, W. C., Jr.: Esophagogastritis complicating aureomycin therapy, New York J. Med. *51:* 1195 (May 1) 1951.
40. Cushing, H.: Peptic ulcers and the interbrain, Surg., Gynec. & Obst. *55:* 1 (July) 1932.
41. Davies, W.: Cardio-chalasia in infancy, J. Pediat. *41:* 467 (Oct.) 1952.
42. Davis, E. D. D.: Anatomy and physiology of cardiac end of the esophagus applied to hiatus hernia and achalasia, J. Laryng. & Otol. *67:* 139 (March) 1953.
43. Day, B. M., and Cunha, F.: Method of increasing gastric reservoir following total gastrectomy, Rev. Gastroenterol. *20:* 705 (Oct.) 1953.
44. Deaton, W. R., and Bradshaw, H. H.: Fate of an isolated segment of the esophagus, J. Thoracic Surg. *23:* 570 (June) 1952.
44a. DeBakey, M. E., Heaney, J. P., and Creech, O.: Surgical considerations in diverticula of esophagus, J.A.M.A. *150:* 1076 (Nov. 15) 1952.
45. DeBakey, M. E., and Ochsner, A.: Surgical treatment of malignant and benign obstructive lesions of esophagus, South. Surgeon *14:* 562 (Aug.) 1948.
46. Dey, F. L., Gilbert, N. C., Trump, R., and Roskelley, R. C.: Reflex shortening of esophagus in experimental animal with production of esophageal hiatus hernia, J. Lab. & Clin. Med. *31:* 499 (May) 1946.
47. Douglas, D.: Modern trends in treatment of dyspepsia, The Medical Press *227:* 500 (May 28) 1952.
48. Effler, D. B., and Collins, E. N.: Complications and surgical treatment of hiatus hernia and short esophagus with thoracic stomach, J.A.M.A. *147:* 305 (Sept. 22) 1951.
49. Effler, D. B., and Ballinger, C. S.: Complications and surgical treatment of hiatus hernia and short esophagus, J. Thoracic Surg. *22:* 235 (Sept.) 1951.
50. Effler, D. B.: Hiatus hernia, Postgrad. Med. *12:* 317 (Oct.) 1952.
51. Efskind, L.: Carcinoma of esophagus, Acta chir. Scandinav. *103:* 401 (Fasc. 6) 1952.
52. Eker, Reidar: Squamous cell carcinomas of esophagus, Acta chir. Scandinav. *103:* 430 (Fasc. 6) 1952.
53. Engel, G. C.: Creation of a gastric pouch following total gastrectomy, Surgery *17:* 512 (April) 1945.
54. Eskridge, M., and Peake, J. D.: Curling of esophagus, South. Med. J. *46:* 213 (March) 1953.
55. Everson, T. C.: Effect of esophagoduodenostomy and esophagojejunostomy on fecal fat and nitrogen loss following total gastrectomy, S. Forum (1951) 1952, pp. 68–71.
56. Farber, S., and others: Case #31311, Mass. Gen. Hosp., New England J. Med. *233:* 162 (Aug. 2) 1945.
57. Feldman, M., and Myers, P.: The co-existence of carcinoma of stomach and esophageal hiatus gastric hernia, Am. J. M. Sc. *224:* 519 (Nov.) 1952.
58. Feldman, M., and Myers, P.: The incidence of co-existence of gastric ulcer in esophageal hiatus hernia of stomach, Rev. Gastroenterol. *20:* 191 (March) 1953.
59. Ferguson, D. J., and others: Studies on experimental esophagitis, Surgery *28:* 1022 (Dec.) 1950.
60. Fincher, E. F., and Swanson, H. S.: Esophageal rupture complicating craniotomy, Ann. Surg. *129:* 619 (May) 1949.
61. Finney, J. M. T., and Rienhoff, W. F., Jr.: Gastrectomy, Arch. Surg. *18:* 140 (Jan.) 1929.
62. Franklin, R. H.: Hiatus hernia in adult, Proc. Roy. Soc. Med. *45:* 289 (May) 1952.
63. Franklin, R. H., and Taylor, S.: Nonspecific granulomatous (regional) esophagitis, J. Thoracic Surg. *19:* 292 (Feb.) 1950.

64. Friman-Dahl, J.: Rotation therapy in cancer of esophagus, Acta chir. scandinav. *103:* 421 (Fasc. 6) 1952.
65. Gardner, W. C., Hartzell, J. B., and Tuttle, W. M.: Simplified technique for treatment of esophageal hiatus hernia, Arch. Surg. *65:* 564 (Oct.) 1952.
66. Geever, E. D., and Merendino, K. A.: An evaluation of esophagitis in dogs following Heller and Grondahl operations with and without vagotomy, Surgery *34:* 742 (Oct.) 1953.
67. Gertz, T. C.: Late results in surgical treatment of achalasia of esophagus, Acta chir. scandinav. *103:* 459 (Fasc. 6) 1952.
68. Gibson, J. B.: Infection of lungs by "Saprophytic" mycobacteria in achalasia of cardia, J. Path. & Bact. *65:* 239 (Jan.) 1953.
69. Gruenwald, P., and Marsh, M. R.: Acute esophagitis in infants, Arch. Path. *49:* 1 (Jan.) 1950.
69a. Harper, R. A. K.: Non-malignant conditions of esophagus, Brit. J. Radiol. *19:* 107 (March) 1946.
70. Harrington, S. W.: Surgical treatment of more common types of diaphragmatic hernia, Ann. Surg. *122:* 546 (Oct.) 1945.
71. Harrington, S. W.: Esophageal hiatus diaphragmatic hernia, Rocky Mountain M. J. *49:* 665 (Aug.) 1952.
72. Hawthorne, H. R., and Davis, H. C.: Surgical methods and results in management of intractable esophageal achalasia, Delaware State M. J. *24:* 306 (Nov.) 1952.
73. Hays, R. P.: Anatomic and physiologic reconstruction following total gastrectomy by use of a jejunal food pouch. S. Forum, 39th Clinical Congress, Amer. Coll. Surg. (1953).
74. Hennessy, R., and others: Esophageal obstruction in childhood, M. J. Australia, *2:* 560 (Oct. 18) 1952.
75. Hoover, W. B.: Esophagitis: Clinical evaluation, Ann. Otol., Rhinol., and Laryngol. *61:* 1148 (Dec.) 1952.
76. Hunt, C. J.: Construction of food pouch from segment of jejunum as substitute for stomach in total gastrectomy, Arch. Surg. *64:* 601 (May) 1952.
77. Hunt, C. J.: Malignant disease of stomach, J. Internat. Coll. Surg. *18:* 651 (Nov.) 1952.
78. Hutton, J. F. K.: Wandering oesophagus, Brit. J. Radiol. *26:* 50 (Jan.) 1953.
79. Isbister, C., and others: Hiatus hernia and stricture of esophagus, M. J. Australia *2:* 355 (Sept. 6) 1952.
80. Ismay, G.: Painful spasm of esophagus, Brit. M. J. *2:* 697 (Sept. 27) 1952.
81. Jackson, C.: Peptic ulcer of esophagus, J.A.M.A. *92:* 369 (Feb. 2) 1929.
82. Janda, C. A., and Van Ravenswaay, A. C.: Regurgitation esophagitis, Arizona Med. *8:* 40 (Jan.) 1951.
83. Jelliffe, A. M., and Ferguson, J. H. L.: Spontaneous rupture of esophagus, Arch. Middlesex Hosp. *3:* 93 (Jan.) 1953.
84. Johnstone, A. S. and others: Non-malignant conditions of esophagus, Brit. J. Radiol. *19:* 101 (March) 1946.
85. Johnstone, A. S.: Radiology of hiatus hernia, Proc. Roy. Soc. Med. *45:* 286 (May) 1952.
86. Jones, A. F.: Diagnosis of hiatus hernia, Proc. Roy. Soc. Med. *45:* 277 (May) 1952.
87. Judd, E. S., and Hoon, J. R.: Total gastrectomy, Arch. Surg. *61:* 102 (July) 1950.
88. Kay, E. B.: Discussion of a paper by: Lam, C. R., and Kenney, L. J.: Problem of hiatus hernia of diaphragm, J. Thoracic Surg. *27:* 12 (Jan.) 1954.
89. Kay, E. B.: Observations as to etiology and treatment of achalasia of esophagus, J. Thoracic Surg. *22:* 254 (Sept.) 1951.
90. Kay, E. B.: Inferior esophageal constrictor in relation to lower esophageal disease, J. Thoracic Surg. *25:* 1 (Jan.) 1953.
91. Kay, E. B., and Brown, K. L.: Partial esophagectomy with end-to-end anastomosis, Surgery *29:* 658 (May) 1951.
92. Kergin, F. G.: Esophageal obstruction due to paraffinoma of mediastinum, Ann. Surg. *137:* 91 (Jan.) 1953.
93. King, A. B., and Reganis, J. C.: Neurogenic erosions of stomach and esophagus, Ann. Surg. *137:* 236 (Feb.) 1953.
94. Kinsella, T. J., Morse, R. W., and Hertzog, A. J.: Spontaneous rupture of esophagus, J. Thoracic Surg. *17:* 613 (Oct.) 1948.
95. Klein, H. C.: Functional tense esophagus and esophageal spasm, Rev. Gastroenterology *19:* 861 (Nov.) 1952.
96. Kleitsch, W. P.: Catastrophic complications of hiatus hernia, Arch. Surg. *65:* 665 (Nov.) 1952.
97. Kline, P. S.: The "gastric cripple" after total gastric resection, J. Internat. Coll. Surgeons *18:* 226 (Aug.) 1952.
98. Koop, C. E., and Johnson, J.: Transthoracic repair of diaphragmatic hernia in infants, Ann. Surg. *136:* 1007 (Dec.) 1952.

99. Laff, H. I.: Cardiospasm associated with pneumonitis, bronchial granuloma and bronchiolithiasis, Ann. Otol., Rhin. & Laryng. *62:* 144 (March) 1953.
99a. Lam, C. R., and Kenney, L. J.: Problem of hiatus hernia of diaphragm, J. Thoracic Surg. *27:* 1 (Jan.) 1954.
100. Leegaard, T.: Corrosive injuries of esophagus, J. Laryng. & Otol. *60:* 389 (Oct.) 1945.
101. Lerche, W.: Esophagus and Pharynx in Action, Springfield, Ill., Charles C Thomas, 1950, p. 216.
102. Levy, H.: Pregnancy complicated by diaphragmatic hernia, J. M. Soc. New Jersey *50:* 72 (Feb.) 1953.
103. Loe, R. H.: Editorial: Importance of esophago-gastric valve mechanism in surgery of stomach and esophagus, Surg., Gynec. & Obst. *94:* 502 (April) 1952.
104. Longmire, W. P., Jr.: Esophageal conditions and their treatment, Am. J. Nursing *47:* 807 (Dec.) 1947.
105. Longmire, W. P., Jr., and Beal, J. M.: Construction of substitute gastric reservoir following total gastrectomy, Ann. Surg. *135:* 637 (May) 1952.
106. Mason, J. M.: Surgical treatment of obstructive lesions of esophagus, Ann. Surg. *127:* 1067 (May) 1948.
107. Mautz, F. R.: Transpleural resections of esophagus and stomach for esophageal obstruction, Ohio M. J. *42:* 1243 (Dec.) 1946.
108. McGlone, F. B.: Total gastrectomy with replacement of stomach by ileocolic segment, J.A.M.A. *151:* 622 (Feb. 21) 1953.
109. McLaren, J. W.: Discussion of a paper: Harper, R.A.K.: Non-malignant conditions of esophagus, Brit. J. Radiol. *10:* 107 (March) 1946.
110. Moersch, H. J.: Hiatal hernia, Ann. Otol., Rhin., & Laryng. *47:* 754 (Sept.) 1938.
111. Moersch, H. J.: Problems in differential diagnosis of lesions of lower portion of esophagus and cardia, Ann. Otol., Rhin., & Laryng. *61:* 976 (Dec.) 1952.
112. Moreton, R. D.: Roentgenologic considerations of gastrointestinal lesions causing hematemesis, South. M. J. *46:* 127 (Feb.) 1953.
113. Moroney, J.: Colonic replacement and restoration of human stomach, Ann. Roy. Coll. Surgeons England *12:* 328 (May) 1953.
114. Mosher, H. P.: Infection as cause of fibrosis of esophagus, Ann. Otol., Rhin., & Laryng. *50:* 633 (Sept.) 1941.
115. Muller, W. H., Byron, F. X., and Power, H. W.: Delayed traumatic rupture of esophagus, J. Thoracic Surg. *25:* 371 (Apr.) 1953.
116. Nicholson, F.: Diaphragmatic hernia, Ann. Surg. *136:* 174 (July) 1952.
117. Nissen, R.: Bridging of esophageal defect by pedicled flap of lung tissue, Ann. Surg. *129:* 142 (Jan.) 1949.
118. Nissen, R.: Surgical diseases of esophagus, Rev. Gastroenterol. *18:* 629 (Sept.) 1951.
119. Nylander, P. E. A., and Elfuing, G.: Some features of esophagus cancer situation in Finland today, Acta chir. scandinav. *103:* 415 (Fasc. 6) 1952.
120. Ochsner, A., and DeBakey, M.: Surgical diseases of esophagus, Laryngoscope *58:* 698 (July) 1948.
121. Olsen, A. M., and Harrington, S. W.: Esophageal hiatal hernias of short esophagus, J. Thoracic Surg. *17:* 189 (April) 1948.
122. Olsen, A. M., Harrington, S. W., Moersch, H. J., and Andersen, H. A.: Treatment of cardiospasm, J. Thoracic Surg. *22:* 164 (Aug.) 1951.
123. Orr, T. G.: Modified technic for total gastrectomy, Arch. Surg. *54:* 279 (March) 1947.
124. Palmer, E. D.: Esophagus and its Diseases, New York, Paul B. Hoeber, Inc., Med. Book Dept. of Harper & Brothers, 1952, pp. 9.
125. Palumbo, L. T., and Brennan, J. E.: Results of total gastrectomy, J. Internat. Coll. Surgeons *17:* 823 (June) 1952.
126. Park, W. D.: Carcinoma of cardiac portion of stomach, Brit. M. J. *2:* 599 (Sept. 13) 1952.
127. Paul, L. W.: Roentgenologic aspects of acute and chronic esophagitis, Radiol. *41:* 421 (Nov.) 1943.
128. Paulson, D. L.: Surgical treatment of esophageal lesions, Arizona Med. *8:* 27 (Nov.) 1951.
129. Penner, A., and Druckerman, L. J.: Segmental spasms of esophagus and their relation to Parkinsonism, Am. J. Digest. Dis. *9:* 282 (Sept.) 1942.
130. Pool, J. L.: Carcinoma of esophagus, diagnostic and treatment problems, Rev. Gastroenterol. *20:* 499 (July) 1953.
131. Potter, W. H., Schaer, S. M., and Stewart, J. D.: Problem of peptic esophagitis, New York J. Med. *51:* 1924 (Aug. 15) 1951.
132. Preiskel, E.: Peptic esophageal ulcer, Lancet (London) *1:* 497 (April 6) 1946.
133. Pringle, J. H., Stewart, L. T., and Teacher, J. H.: Digestion of esophagus as cause of postoperative and other forms of hematemesis, J. Path. & Bact. *24:* 396 (Oct.) 1921.
134. Puestow, C. B.: Carcinoma of cardiac end of stomach and esophagus, Minnesota Med. *36:* 170 (Feb.) 1953.

135. Puppel, I. D.: Role of esophageal motility in surgical treatment of mega-esophagus, J. Thoracic Surg. *19:* 371 (March) 1950.
136. Rall, J. E., Gilbert, N. C., and Trump, R. C.: Effect of vagus stimulation on longitudinal fibers of stomach and esophagus, Quart. Bull., Northwestern Univ. M. School *19:* 194, 1945.
137. Ransom, H. K.: Total gastrectomy, Arch. Surg. *55:* 13 (July) 1947.
138. Ratjen, E.: Concomitant arterio-mesenteric obstruction of duodenum and chronic oesophagitis, Acta radiol. *37:* 369 (March–April) 1952.
139. Rector, L. E., and Connerley, M. L.: Aberrant mucosa in esophagus in infants and in children, Arch. Path. *31:* 285 (March) 1941.
140. Richards, L., and Dietrich, H. J., Jr.: Blind bouginage in treatment of benign esophageal obstruction, Ann. Otol., Rhin., & Laryng. *61:* 1134 (Dec.) 1952.
140a. Rigler, L. G., and Eneboe, J. B.: The incidence of hiatus hernia in pregnant women and its significance, J. Thoracic Surg. *4:* 262 (Feb.) 1935.
141. Ripley, H. R., and others: Experimental studies of peptic ulceration and stricture of lower part of esophagus, Forum (1950), p. 60, 1951.
141a. Ripley, H. R., Olsen, A. M., and Kirklin, J. W.: Esophagitis after esophagogastric anastomosis, Surgery *32:* 1 (July) 1952.
142. Rives, J. D., and Strug, L. H.: Surgical repair of esophageal hiatus hernia, Ann. Surg. *115:* 745 (May) 1942.
143. Robertson, R., and Sarjeant, T. R.: Reconstruction of esophagus, J. Thoracic Surg. *20:* 689 (Nov.) 1950.
144. Rosenberg, N., Kunderman, P. J., Vroman, L., and Moolten, S. E.: Prevention of experimental esophageal stricture by cortisone, Arch. Surg. *63:* 147 (Aug.) 1951.
145. Rosenberg, N., Kunderman, P. J., Vroman, L., and Moolten, S. E.: Prevention of experimental esophageal strictures by cortisone, Arch. Surg. *66:* 593 (May) 1953.
146. Rotter, W., and Hasse, G.: On a case of chronic peptic esophagitis, Zentralbl. allg. Path. *85:* 167 (June 10) 1949.
147. Rubin, J.: Congenital defect of diaphragm with unusual features, Brit. J. Radiol. *26:* 154 (March) 1953.
148. Sanford, M. C.: Congenital diaphragmatic hernia, Clin. Proc. Child. Hosp. *8:* 252 (Nov.) 1952.
149. Scannell, J. G.: Thoracic surgery, New England J. Med. *247:* 843 (Nov. 27) 1952.
150. Scannell, J. G. and others: Case #34462, Mass. Gen. Hosp., New England J. Med. *239:* 752 (Nov. 11) 1948.
151. Schalm, L.: Late and fatal complication after oesophago-gastrostomy (operation of Heyrovsky), Arch. chir. neerl. *2:* 166, 1950.
152. Scheel, A.: Results of radiation treatment of esophageal cancer at Det Norske Radium hospital, Acta chir. scandinav. *103:* 425 (Fasc. 6) 1952.
153. Scott, M. G.: Gastro-colic fistula with reflux into esophagus, Brit. J. Radiol. *26:* 268 (May) 1953.
154. Selye, H.: Experimental production of peptic hemorrhagic esophagitis, Canad. M.A.J. *39:* 447 (Nov.) 1938.
155. Seybold, W. D.: Carcinoma of esophagus; surgical treatment, Texas J. Med. *49:* 214 (April) 1953.
156. Shoulder, H. H.: Esophageal leiomyoma: surgical treatment, Missouri M. *50:* 49 (Jan.) 1953.
157. Smithers, D. W.: Short esophagus and its association with peptic ulceration and cancer, Brit. J. Radiol. *18:* 199 (July) 1945.
158. Steiner, G.: Chronic ulcerative esophagitis with report of case of ulcer in esophageal varices, Brit. J. Radiol. *19:* 145 (April) 1946.
159. Stewart, M. J., and Hartfall, S. J.: Chronic peptic ulcer of esophagus, J. Path. & Bact. *32:* 9 (Jan.) 1929.
160. Strieder, J. W.: Medical progress: surgery of esophagus, New England J. Med. *243:* 445 (Sept. 21) 1950.
161. Strothers, H. H.: Chemical burns and strictures of esophagus, Arch. Otolaryng. *56:* 262 (Sept.) 1952.
162. Sweet, R. H.: Esophageal hiatus hernia of diaphragm, Ann. Surg. *135:* 1 (Jan.) 1952.
163. Sweet, R. H.: Analysis of 130 cases of hiatus hernia treated surgically, J.A.M.A. *151:* 376 (Jan. 31) 1953.
164. Sweet, R. H.: Discussion of a paper: Kay, E. B.: Observations as to treatment of achalasia of esophagus, J. Thoracic Surg. *22:* 254 (Sept.) 1951.
165. Sweet, R. H.: Peptic ulceration of cardia and lower esophagus, Postgraduate Course, Surgery of the Upper Gastrointestinal Tract, Clinical Congress of the Amer. Coll. Surg. (Oct. 8) 1953.
166. Sweetser, H. B.: Hiatus hernia masquerading as coronary disease, Minnesota Med. *35:* 971 (Oct.) 1952.

167. Szilagyi, D. E., Connell, T. H., Jr., and Fallis, L. S.: Observations on transposition of the ileocolic segment as a food pouch after total gastrectomy, S. Forum (1951), 1952, pp. 68.
168. Tanner, C. H.: Carcinoma of esophagus, The Medical Press, *229:* 33 (Jan. 14) 1953.
169. Therkelsen, F.: Esophageal resection by right sided approach, Acta chir. scandinav. *103:* 418 (Fasc. 6) 1952.
170. Thorek, P.: Surgical treatment of stenosis due to esophagitis, J.A.M.A. *147:* 640 (Oct. 13) 1951.
171. Valdoni, P.: Radical treatment of esophageal stenosis, La Presse med. *59:* 1216 (Sept. 19) 1951.
172. Van Aken, H.: Stenosing peptic esophagitis, Arch. chir. neerl. *2:* 153, 1950.
173. Vinson, P. P.: Management of patients suffering from difficulty in swallowing, Postgrad. Med. *12:* 393 (Nov.) 1952.
174. Vinson, P. P.: The Diagnosis and Treatment of Diseases of the Esophagus, Springfield, Ill., Charles C Thomas, 1940.
175. Vinson, P. P., and Butt, H. R.: Esophagitis; clinical study, J.A.M.A. *106:* 994 (Mar. 21) 1936.
176. Wandall, H. H.: Oesophagitis-oesophageal ulcer, aetiology and pathogenesis, Acta med. scandinav. Supplement 262–266, p. 947, 1951–52.
177. Wangensteen, O. H.: Discussion of a paper: Nissen, R.: Surgical diseases of esophagus, Rev. Gastroenterol. *18:* 629 (Sept.) 1951.
178. Wangensteen, O. H.: Discussion of a paper: Kay, E. B.: Observations as to treatment of achalasia of esophagus, J. Thoracic Surg. *22:* 254 (Sept.) 1951.
179. Wagensteen, O. H., Merendino, K. A., and Varco, R. L.: Displacement of esophagus into a new diaphragmatic orifice in repair of paraesophageal and esophageal hiatus hernia, Ann. Surg. *129:* 185 (Feb.) 1949.
180. Wangensteen, O. H.: Physiologic operation for mega-esophagus, Ann. Surg. *134:* 301 (Sept.) 1951.
181. Wangensteen, O. H.: An assessment of etiologic aspects of peptic ulcer and surgical therapy, Tr. & Stud. Coll. Physicians Philadelphia, *18:* 1 (April) 1950.
182. Wangensteen, O. H., and Leven, N. L.: Gastric resection for esophagitis and stricture of acid-peptic origin, Surg., Gynec. & Obst. *88:* 560 (May) 1949.
183. Ware, B. I., Shnider, B. I., and Davis, E. W.: Spontaneous rupture of esophagus, Arch. Surg. *65:* 723 (Nov.) 1952.
184. Welin, S.: Contribution to roentgen diagnostics of limited esophagitis, Acta radiol. *27:* 461 (Fasc. 4) 1946.
185. West, J. P.: Total gastrectomy for carcinoma of stomach, Ann. Surg. *129:* 373 (March) 1949.
186. West, J. P. and Fenger, J. R.: Results of total gastrectomy for cancer of stomach, Ann. Surg. *135:* 497 (April) 1952.
187. Wilson, E.: Acute oesophagitis, M. J. Australia, *1:* 820 (June 14) 1952.
188. Wilson, J. L.: Stenosing peptic esophagitis, Laryngoscope, *61:* 423 (May) 1951.
189. Winkelstein, A.: Peptic esophagitis, J.A.M.A. *104:* 906 (March 16) 1935.
190. Wolf, S., and Wolff, H. G.: The gastric mucosa, "Gastritis" and ulcers, Am. J. Digest. Dis. *10:* 23 (Jan.) 1943.
191. Womack, N. A., Brintnall, E. S., and Ehrenhaft, J. L.: Benign obstruction of lower esophagus, J.A.M.A. *145:* 283 (Feb. 3) 1951.
192. Wooler, G. H.: Treatment of cardiospasm, Brit. M. J. *2:* 100 (July 9) 1949.
193. Wooler, G. H.: Mechanism of cardia, Proc. Roy. Soc. Med. *45:* 290 (May) 1952.

# AN OPERATION FOR ABSCESS OF THE APPENDIX VERMIFORMIS CAECI*

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Familiar as we are with the diseases to which the alimentary canal is liable, there is one part of it—the appendix vermiformis—the pathology of which is, to a great extent, involved in obscurity. In the ancient literature of the profession we find nothing in regard to it; in fact, we have no reason to suppose that attention was ever directed to it as a part which might be the seat of special disease. And this neglect is not to be wondered at, when we reflect that, by its position, the appendix vermiformis is insignificant; and, by its anatomical similitude with the rest of the intestinal tube, it is impossible for disease affecting it to remain for more than a limited period, local. In many cases, general symptoms too rapidly intervene to allow an exact diagnosis to be made out; and, frequently, it is only at the autopsy that we become acquainted with the precise nature of the disease which has terminated the life of the patient.

During the present century, however, the zeal of the profession, which has been at work, uprooting the hidden things of the past, has not suffered investigation into the obscure physiology and pathology of the appendix, to remain wholly idle. Various papers, by English, French and German authors, have appeared;[1] besides occasional cases, which from time to time have found their way into the medical journals. But although much has already been done, much still remains to be accomplished, before the obscurity surrounding this subject will have been entirely removed.

There are, however, three diseases of the appendix vermiformis, which have been classified from post-mortem appearances:

1. Gangrene.
2. Perforating Ulcer.
3. Abscess.

* *From the Medical Record, Volume 2. March 1, 1867–February 15, 1868. Original Communications.*

[1] Husson and Dance, "Sur quelques Engorgements Inflammatoires qui se developpent dans la Fosse Iliaque Droite. Repertoire General. Tome iv., 1827." "Des Abces de la Fosse Iliaque Droit. Lecons Orales de Dupuytren. Tome iii., 1833." By these observers, abscesses of the right iliac fossa, and the symptoms accompanying them, were described, but they failed to perceive the connexion which these bore to inflammations originating in the appendix vermiformis. They thought that they were all "primarily inflammations of the cellular tissue in the right iliac fossa, which may terminate in resolution, suppuration, or universal peritonitis." It was reserved for Dr. Burne, "Medico-Chirurgical Transactions," vols xx and xxii., to establish the true relations of these abscesses, and trace them back from effect to cause.

To these, Rokitanski adds a catarrhal affection of the appendix, which may be only the commencement of a graver condition, or which may exist by itself as a distinct disease. By some, these diseases have all been grouped together under the general name, Typhlitis.[2] They all have the same origin, viz. the impaction of calculous masses in the calibre of the tube, or the presence of foreign substances, such as the seed of fruit, shot, etc. These may become causes of disease, either by obstructing the circulation of the part, or by their presence, simply, they may give rise to irritation and inflammation.

Gangrene, and perforating ulcers of the appendix vermiformis, usually occur in male children, and prove fatal in from two to five or six days. No certain diagnosis can be made out. Violent symptoms of general peritonitis set in, and it is rare that medical interference is of any avail.

Abscess of the appendix, on the contrary, generally attacks those past adult age; is slower in its progress, and presents symptoms more easy to recognize. An attack is ushered in by pain in the bowels, suddenly coming on; loss of appetite; nausea, vomiting, and perhaps some febrile action. In a short time the pain circumscribes itself in the right iliac region; nausea and vomiting continue; the bowels are constipated; skin dry and feverish; pulse inflammatory; tongue thickly coated; abdomen somewhat tumid and resonant on percussion—also sympathetic tenderness over its whole extent. After the lapse of a few days, a small circumscribed tumor may be detected in the neighborhood of the right iliac fossa.

All of the symptoms may not be present in every case, and some of them are common to other diseases. Uncertainty may exist with regard to the diagnosis. It may be difficult to determine whether a case is internal hernia, abscess of the appendix, or intussusception. The greatest diagnostic mark to be remembered, is this: In the above-named diseases (internal hernia and intussusception) there is insuperable obstruction in the bowels, while in abscess of the appendix vermiformis there is no such obstruction. The administration of a cathartic, then, will clear up all obscurity. And it is to be remembered, also, that in abscess of the appendix, the action of a cathartic produces, at first, marked relief of all the symptoms; but this relief is only temporary. Accordingly, then, as a cathartic acts efficiently or not at all, we are able, by exclusion, to arrive at a tolerably certain diagnosis; the more so, as we are aided by the symptoms already enumerated, the pain and circumscribed tenderness over the iliac fossa.

The prognosis is bad. The end is generally death. Dr. Lewis reports[3] forty-seven cases of diseased appendix, all of which terminated fatally. Death occurs in this way: When the pus has accumulated in sufficient quantity, it forces its way into the cavity of the peritoneum, a violent peritonitis is induced, and death rapidly ensues. All, however, who have abscess of the appendix vermiformis, do not die. In a few cases, adhesions, strong enough to keep the contained pus from invading the peritoneal cavity, are created around the abscess and the pus finds an exit by ulceration, either into some portion of intestine, generally the caecum, or through the abdominal wall.

[2] Purchelt, the first German physician who gave particular attention to these conditions, proposed the name perityphlitis.

[3] New York Journal of Medicine, November, 1856.

The following cases are illustrative:

In 1843, I was called in consultation to visit Dr. T., of Brooklyn. He had been confined to bed for some weeks, suffering from pain in the bowels, constipation, disturbance of system, fever, tenderness in right inguinal region, etc. On examination, I found a swelling in the neighborhood of the iliac fossa, in which questionable fluctuation existed. An opening of exploration was made, which justified a free incision. I accordingly cut down into, and excavated the contents of the abscess; with the pus, a little concretion, the size of a raisin-seed, came out. In a short time the patient recovered, and is living now, in good health.

I saw Mr. D., aet. forty-five, a stong, healthy farmer, on the 20th of May, 1865. He had been sick for six days with pain in the bowels, tenderness over right iliac fossa, etc. He was lying on his back, his legs drawn up; his bowels had been freely moved. I prescribed a blister, opium, and nourishment. I next saw him on the 17th of June. He was weak and emaciated from the severity and continuance of the attack. A tumor had appeared over the region of tenderness, which two days previously had grown considerably smaller, and at the same time a liquid, supposed to be purulent, discharge had taken place from the bowels. The tumor was soft to the touch, and presented a point where pus was near the surface: this I laid open with a bistoury, and considerable purulent matter, with gas, made its escape. Following this was improvement and perfect recovery. In this case the abscess had broken, not only through into the intestine, as was shown by the reduction in the size of the tumor, simultaneously with a discharge from the bowels, but ulceration was already commencing to form a track for the escape of the pus through the abdominal walls.

C. J. O., aet. twenty-one, robust, had suffered for two years from dyspepsia and chronic diarrhoea. He stated that, on the 10th of June, 1865, he was attacked with peritonitis, the pain being intensified over the right iliac fossa. On the 16th, the general inflammation having abated, he noticed a swelling, hard to the touch, and very painful, above the right groin. This tumor gradually enlarged, until the 20th of August, when I first saw him. It presented a circumscribed appearance, had a doughy feel, and sometimes crepitated as if it contained gas. Leeches were applied and poultices; the bowels were made to act, and he had got so far well as to be able to attend to his business, though the tumor and hardness had never entirely disappeared; when, in the latter part of November, he had a recurrence of his difficulty. On the 4th of December I made an incision, through which some pus and a small calculus made their escape. On examination the latter was found to consist of uric acid, biliary matter, and some debris of muscular food. On the 16th of January, the tumor in the meantime having disappeared, leaving only an indurated hardness, the patient was again attacked with violent pain, vomiting, etc., as at the first. On the 17th, some enlargement was noticed, and on the 20th (it having attained the size of a man's fist, and fluctuation being discovered), it was opened, and pus copiously discharged therefrom. In a short time the flow of pus had ceased, the parts resuming very nearly their normal condition. He soon returned to his home in Connecticut, where he now

is. In a letter dated April 6, 1866, he says that his general health is poor, being troubled with weak bowels and vomiting, but his side has given him no inconvenience since it was last opened.

A fact, which bears somewhat upon the prognosis, is discovered in looking at the anatomical position of the appendix. Its origin is generally from the inferior, posterior, and inner side of the caecum, and it may be of varying size, and occupy various positions. It may depend into the pelvis and lie over the iliac fossa; it may lie curled up behind the caecum, on the outer edge of the psoas magnus, upon the fascia iliaca; or it may lie encircling the caecum and extend up along the colon for four or five inches. By remembering the last position our diagnosis will be aided, rather than the prognosis, for we will be prepared to find the tenderness and circumscribed swelling at a higher point upon the abdomen, than the region over the iliac fossa. When the appendix lies behind the caecum upon the fascia iliaca, its inflammation and abscess become of less importance, for the peritoneum in this case is not involved, and the direction of the abscess is towards the outer margin of the quadratus lumborum, where the parietes offer but little resistance. These last positions of the appendix, however, are rare, and they are only mentioned as being among the possibilities.

The duration is variable, and depends upon the absence or presence of adhesions. In the first case the patient dies in from two to four or five days. In the second, death cannot occur so long as the adhesions keep the peritoneal cavity intact. These are in danger of being broken as long as the formation of pus is on the increase; that is, until its maximum is reached, which, in all probability, will be by the twelfth day. The adhesions, if they break at all, will have broken by this day, so that we may conclude, if the patient survives the fifth day, Nature is putting her seal of safety upon the case. Death may occur after the twelfth day, but the chances are greatly diminished.

Of the general treatment I have little to say. Absolute rest on the back, opium, and such nourishment as will maintain the plastic integrity of the blood, will be sufficiently indicated to every practitioner.

The matter of local treatment, however, has attracted my attention for many years. These questions presented themselves: Are the efforts of nature exerted in behalf of a cure, and if so, in what way? Observation indicates the reply and experience verifies its truth. Nature does labor in behalf of life, in two ways: 1st, by means of the wall of false membrane, which she builds up around the abscess; and 2d, by the ulceration, which gives an external vent to the escape of its contents. This being settled, it became a question whether surgery might be able to render assistance to Nature in this work; and if so, at what period would interference best come in?

In what has gone before, it would seem that in some cases no adhesions are formed, and general peritonitis quickly carries the patient to the grave. It is obvious, in these cases, that surgery can be of no avail.

It has likewise been seen that many cases are prolonged beyond the fifth day. In these we have evidence of an adhesive inflammation, and we know that the

next step in the progress of the case will be ulceration. The only danger now to be feared is, that before ulceration can fully accomplish its purposes, the pressure of the accumulating pus will break through the recently formed adhesions, and the event will be the same as if no adhesive inflammation had taken place. Just here, if at all, the office of the surgeon becomes necessary; for by making a free incision down to the point where pus is collected, we have a method by which we may supersede nature in its slow process of ulceration. By this means, the pus will escape as soon as it is formed; consequently there will be no pressure upon the recent adhesions, and even though weak, they will be strong enough to answer all the purposes for which they were created. The time for making the incision is a matter of great importance. To be successful, it is necessary that it should be made neither too early nor too late—not before adhesions are fully formed, nor after a short period before the maximum formation of pus has been reached; that is, the incision should be made after the fifth day, and before the twelfth.

Reasoning thus, I had convinced myself of the practicability of an operation, in cases of abscess of the appendix vermiformis. Of its safety there could be no doubt, for there was no danger in the division of those structures through which the incision would pass. That it was perfectly justifiable, I had no doubt, for, taking into consideration the two possibilities of any case, I could find no reason why the operation should not be performed. The first of these, was where the diagnosis had been clearly made out. This has been sufficiently dwelt upon in the foregoing paragraphs. The second, was in those cases in which there was doubt as to the diagnosis. If no abscess had already formed, in case one should be in process of formation, an external opening would tend to make it point in a safe direction. And even if no abscess should form, a free incision would relieve tension, thus adding to the comfort of the patient, and in no way prejudicing his safety. One other question remained. Would the operation be successful in bringing about a cure? Judging from the result of the three cases reported above, an affirmative answer seemed certain; for these recovered, because in each one nature had provided for an external discharge of the contents of the abscess, and what nature had provided for in these three, an operation would provide for in all cases. These theories received final confirmation in the result of the case, the report of which follows:

J. D., aet. 40, strong, a full habit, was attacked with pain in the bowels at half-past four o'clock in the morning of Friday, January 12, 1866. He has been more or less subject to severe attacks in the bowels for some years. The night preceding the present attack, he had been out with friends and had eaten a late supper. At five A.M. he vomited, and felt some pain in the right iliac fossa. At eight, he took coffee and toast, after which he went down town. During the day he had no appetitite, and took no food until evening, when he ate a little cold chicken. There was no movement from the bowels during the day. In the night he had nausea. On the 13th he arose early, having passed a restless night. After breakfast he took two blue-pills. During the day he suffered from pain in the

right side, which was relieved by bending over to that side. On the 14th, there being as yet no movement from the bowels, he took a glass of Saratoga Empire water. Pain, restlessness, and nausea still continued. Mustard was applied over the affected side. This evening he had an inefficient movement from the bowels. On the 15th, the above symptoms increased in severity, continued, and in addition he was troubled with eructations. In the evening his family physician, Dr. Sabine, saw him, and ordered opium and a blister to the affected side, also leeches. On the 16th, I saw the patient in consultation with Dr. S. We found him with thickly coated tongue; no appetite; nausea, and a constipated state of the bowels; a pulse inflammatory, ranging over 100; skin dry and feverish; abdomen tumid and resonant on percussion; pain in the whole abdomen, but more decided in the right iliac fossa. Over this was a circumscribed tenderness, the boundaries of which could easily be marked out by the fingers. Micturition painful; pain also extended down the right thigh, and to the right testicle, which was drawn up. He was lying with his right thigh flexed on the pelvis, which position gave him some relief.

The case was diagnosed as probably abscess of the appendix vermiformis. To render it certain, however, that there was no internal hernia, intussusception, or impaction of faeces, and to clear up the diagnosis, we ordered calomel, gr. xv., and opium, gr. iij., to be given in three doses. On the morning of the 17th, ol. ricini ℥ j, with tr. opii gtt. xx., were administered, and operated freely, a large quantity of scybalous faeces coming away. The immediate effect was an amelioration of all the symptoms. The pulse became soft, and fell to 80; skin moist; tongue less coated; some return of appetite; abdomen less tumid, and the pain became definitely circumscribed in the right iliac fossa. On the 19th, his symptoms indicated a return of his former condition. During this day he was kept under the influence of opium, and on the 20th, finding his symptoms more unfavorable, his exact condition was explained to him and the operation proposed. It was, with his consent, decided that if on the next day there should be no improvement, it should be performed. On the 21st, there being no change for the better, but if anything for the worse, and it now being the ninth day of the attack, it was decided to operate as soon as practicable. An injection of catnip tea was given, to relieve the bowels of wind, and at half-past two P.M., assisted by Drs. Sabine, Sands, Thomas Sabine, and my pupil, Mr. Wynkoop, I commenced the operation.

An incision six inches in length was made through the integument, commencing above, and about one inch from the anterior superior spinous process of the ilium, running towards the symphysis pubis. About one inch of the incision was above an imaginary line drawn from one ant. sup. spin. proc. to the other, and five inches below. The incision was continued carefully down, and all the structures found to be healthy, until the fascia transversalis was reached, which was found to be thickened. This was divided over a director, and right beneath a tumor was felt, which was about two inches long and an inch and a half in width. An exploring needle was introduced, when immediately there gushed up some

thick, bad-smelling pus. The sac was now freely opened, and about four ounces of pus, in which there may have been a little faeces, discharged. A tent was introduced into the cavity, and the wound left to close up by granulations. The patient rallied well, after the operation, and passed a good night. The next morning he was in a quiet condition; pulse 84, soft; tongue more moist; abdomen soft; but little fever; wind escaping freely from the bowels; wound discharging healthily. The after treatment consisted entirely of rest, opium, and nourishment. Perfect recovery took place in three weeks, and at the date of writing (December, 1866) he is enjoying perfect health.

## BOOK REVIEWS

The editors of THE AMERICAN SURGEON will at all times welcome books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.

*Neurosurgery of Infancy and Childhood.* By FRANK D. INGRAHAM, M.D., Associate Professor of Surgery, Harvard Medical School, Neurosurgeon-in-Chief, Children's Medical Center, Neurological Surgeon, Peter Bent Brigham Hospital, Boston, Massachusetts, and DONALD D. MATSON, M.D., Assistant Professor of Surgery, Harvard Medical School, Neurosurgeon, Children's Medical Center, Senior Associate in Neurological Surgery, Peter Bent Brigham Hospital, Boston, Massachusetts. Charles C Thomas, Springfield, Illinois, 1954. 443 pages. $15.00.

The vast amount of case material that passes through the neurosurgical service of the Children's Medical Center in Boston makes it an ideal source of data for a treatise on neurosurgery of infancy and childhood. The authors have beautifully compiled this rare experience into a concise yet complete book which has been eagerly awaited in neurosurgical circles.

The 380 excellent illustrations aid greatly this book's readability. All things pertinent to the difference in diagnosis and treatment of childhood from adult neurosurgical problems are stressed, and the meticulous attention to details necessary in the management of infants is well emphasized. The excellent results and low mortality rates presented are a proper tribute to the skill and devotion of the authors to this challenging field, which all too often has been called depressing.

Neurosurgeons whose pediatric experience is of necessity small will find invaluable information here, and the book's organization, clarity, and freedom from superfluous verbiage enhances its value to the pediatrician and general surgeon. This volume will undoubtedly retain a permanent prominent niche in neurosurgical literature.

WILLIAM P. WILLIAMSON, M.D.

*Diseases of the Knee. Management in Medicine and Surgery.* By ANTHONY F. DEPALMA, M.D., James Edward Professor of Orthopedic Surgery and Head of the Department, Jefferson Medical College, Philadelphia; Attending Orthopedic Surgeon, Jefferson Medical College Hospital, Philadelphia; Attending Orthopedic Surgeon, Methodist Episcopal Hospital, Philadelphia; Chief Orthopedic Surgeon (Rotating), Philadelphia General Hospital; Consultant Orthopedic Surgeon, New Jersey Orthopedic Hospital; Consultant Orthopedic Surgeon, Veterans Hospital, Philadelphia. J. B. Lippincott Company, Philadelphia-London-Montreal. 455 Illustrations. Drawings by Carl Brill. $20.00.

This volume of 840 pages with 455 illustrations provides a complete and comprehensive coverage of diseases of the knee. Anyone familiar with the author's previous book on the shoulder will readily understand how this voluminous coverage of such a limited segment of the body as the knee can be presented without undue repetition or inconsequential details.

The first sections of the book are devoted to the anatomy and mechanics of the knee joint with an interesting discussion of the evolution of the pelvic girdle and lower extremities from the primitive fishes through the amphibians, lower mammals and man. There is also a discussion with excellent illustrations of the comparative anatomy of the frog, turtle, opossum, dog, monkey and other animals with an analysis of the variations found based upon the differences of posture and mechanical function.

The second and largest portion of the book concerns the various disabilities and diseases occurring about the knee. The sections on traumatic lesions of the menisci and ligaments

and on disorders of the extensor apparatus are especially well presented. The detailed discussion of the arthritides in a work of this type might seem superfluous, however, it does provide an ample background for understanding the outlined management of arthritis as it involves the knee.

As the various disabilities and diseases are discussed, surgical procedures aimed at their correction or care are outlined. In addition there is an entire section devoted to surgical approaches and procedures including amputation above, through and below the knee. The final chapter deals with bone neoplasms as they involve the region of the knee joint.

This volume is beautifully put together and the illustrations are excellent. Each chapter is followed by an extensive bibliography for those who may wish to delve even further into the subject matter presented. The work will serve as a superb reference for anyone treating patients with afflictions of the knee.

GEORGE A. HIGGINS, M.D.

## BOOKS RECEIVED

Books received are acknowledged in this section, and such acknowledgement must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

*Mayo Clinic Diet Manual*. Second Edition. By The Committee on Dietetics of the Mayo Clinic, W. B. Saunders Company, Philadelphia, London, 1954.

*Current Therapy 1954. Latest Approved Methods of Treatment for the Practicing Physician*. Edited by HOWARD F. CONN, M.D. Consulting Editors: M. EDWARD DAVIS, VINCENT J. DERBES, GARFIELD G. DUNCAN, HUGH J. JEWETT, WILLIAM J. KERR, PERRIN H. LONG, H. HOUSTON MERRITT, PAUL A. O'LEARY, WALTER L. PALMER, HOBART A. REIMANN, CYRUS C. STURGIS, ROBERT H. WILLIAMS, W. B. Saunders Company, Philadelphia and London.

*Atlas of Orthopedic Traction Procedures*. By CARLO SCUDERI, B.S., M.D., M.S., Ph.D. Clinical Associate Professor of Surgery, University of Illinois; Professor of Surgery, Cook County Graduate School; Attending Surgeon, Cook County Hospital; Chairman of Department of Orthopedic Surgery, St. Elizabeth's Hospital and Columbus Hospital; Senior Orthopedic Surgeon, Alexian Brothers' and St. Anne's Hospitals; Consulting Orthopedic Surgeon, Augusta Hospital, Chicago; Consulting Orthopedic Surgeon, McNeal Memorial Hospital, Berwyn, Illinois; Member of American Academy of Orthopedic Surgery, Clinical Orthopedic Society, American Association for the Surgery of Trauma, Western Surgical Association, American Medical Association, American College of Surgeons. With 124 Illustrations. St. Louis. The C. V. Mosby Company. 1954. $12.50.